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A STUDY OF THE ROENTGENOLOGIC APPEARANCE OF THE LOBES OF THE LUNG AND THE INTERLOBAR FISSURES[1]

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ROENTGENOGRAMS consist of superimposed shadows of a three-dimension object projected on a flat plane. The shadows obtained on the film are of two dimensions and do not give a true picture of the shape of the process or its location. To bring out the third dimension, stereo-roentgenograms are often taken. The use of postero-anterior and lateral projections gives us two films in planes at right-angles to each other, which is of great aid in studying the position and shape of the parts under consideration.[2]

The necessity of knowing the exact position and shape of a disease process is especially important in the chest. Processes occurring in the interlobar septa offer particular difficulty in interpretation on a single postero-anterior projection. Because of the oblique position of the septa, the horizontal ray projects the shadow of the septa on the roentgenogram as a broad surface, giving the impression of a diffuse area of lung involvement. Stereo-roentgenograms do not bring out the true nature or location of this process. A lateral view, in the same plane as the interlobar fissure, gives us a totally different conception as to the limits of the process.

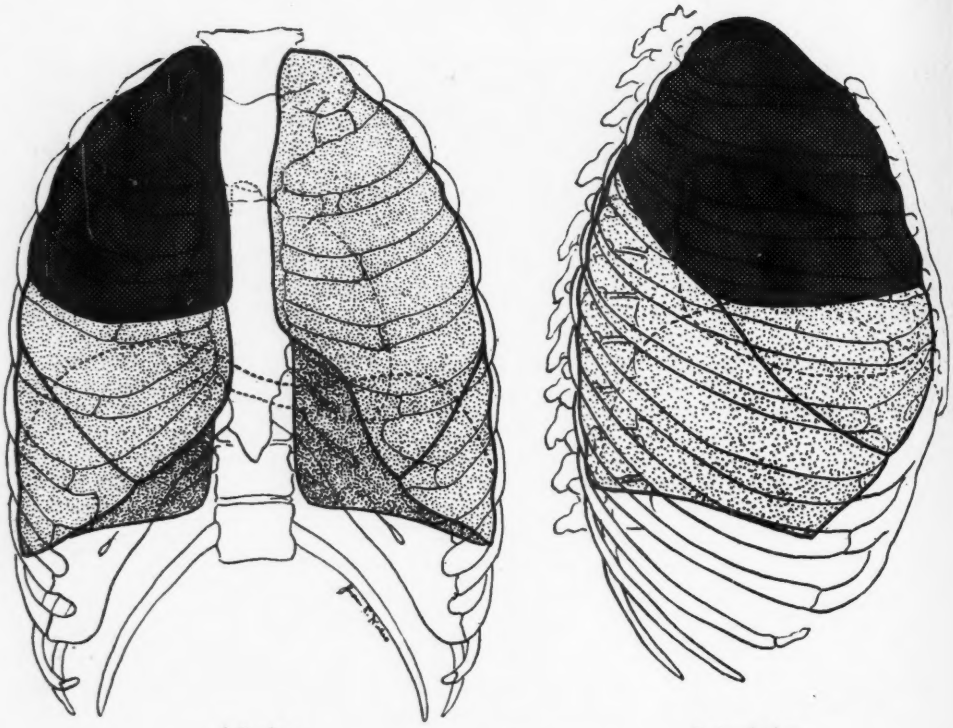
No longer should roentgenologists be satisfied to make a diagnosis of shadows of increased density occupying the upper or lower part of the chest. With the demand for information made by the chest surgeon, roentgenologists are called upon to be more accurate in locating the disease process, as well as to be more specific regarding its nature.

Considerable literature has appeared concerning the roentgenologic appearance of certain disease processes, but there is no satisfactory study available as a guide to the exact appearance on the roentgenogram of the lobes of the lung and the interlobar septa.

This study was undertaken to provide such a set of diagrams, showing the appearance of the lobes of the lung and interlobar septa in the postero-anterior and lateral views. The shadows obtained were subsequently compared to clinical cases of unquestioned diagnoses. We do not mean to imply that all disease processes affecting these divisions of the lung would occupy the areas designated in the diagrams: previous disease conditions of the lung may have resulted in retraction, distortion, or displacement of the region of the lung under consideration. The entire interlober septum may not be, and usually is not, involved by the disease process. Likewise, only a portion of one or more

[1] Supported by the Lily Spreckels Weggeforth Fund.

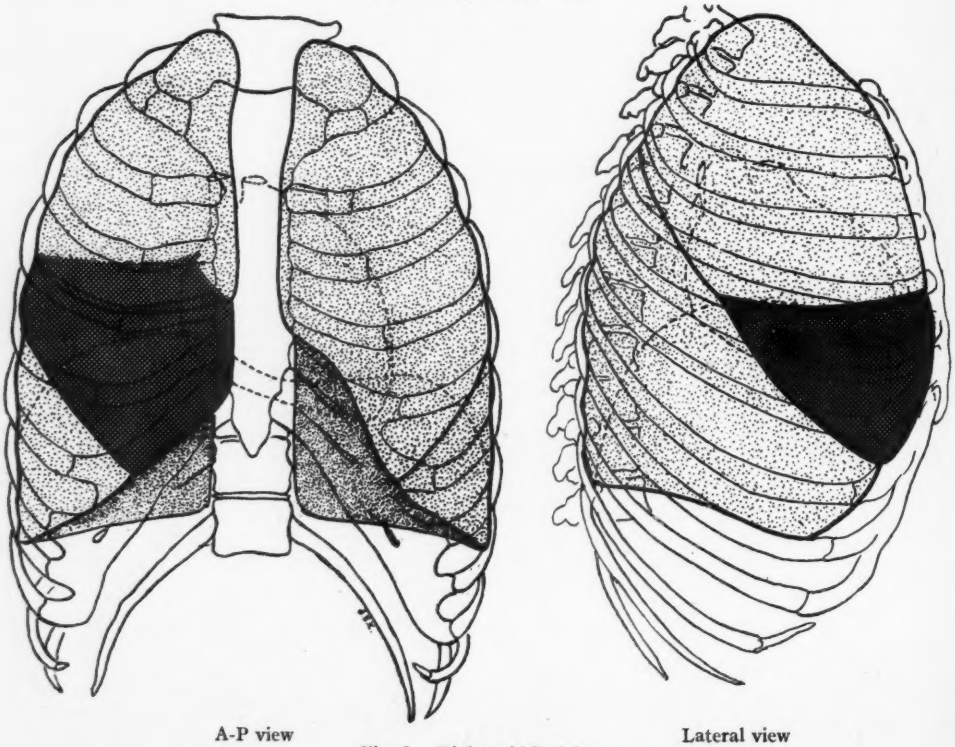
[2] The reader will observe that the illustrations in this paper do not occur in numerical order: this is due to the unusual number and large size of the illustrations, coupled with the terse style of the text.



A-P view

Lateral view

Fig. 1. Right upper lobe.



A-P view

Lateral view

Fig. 2. Right middle lobe.

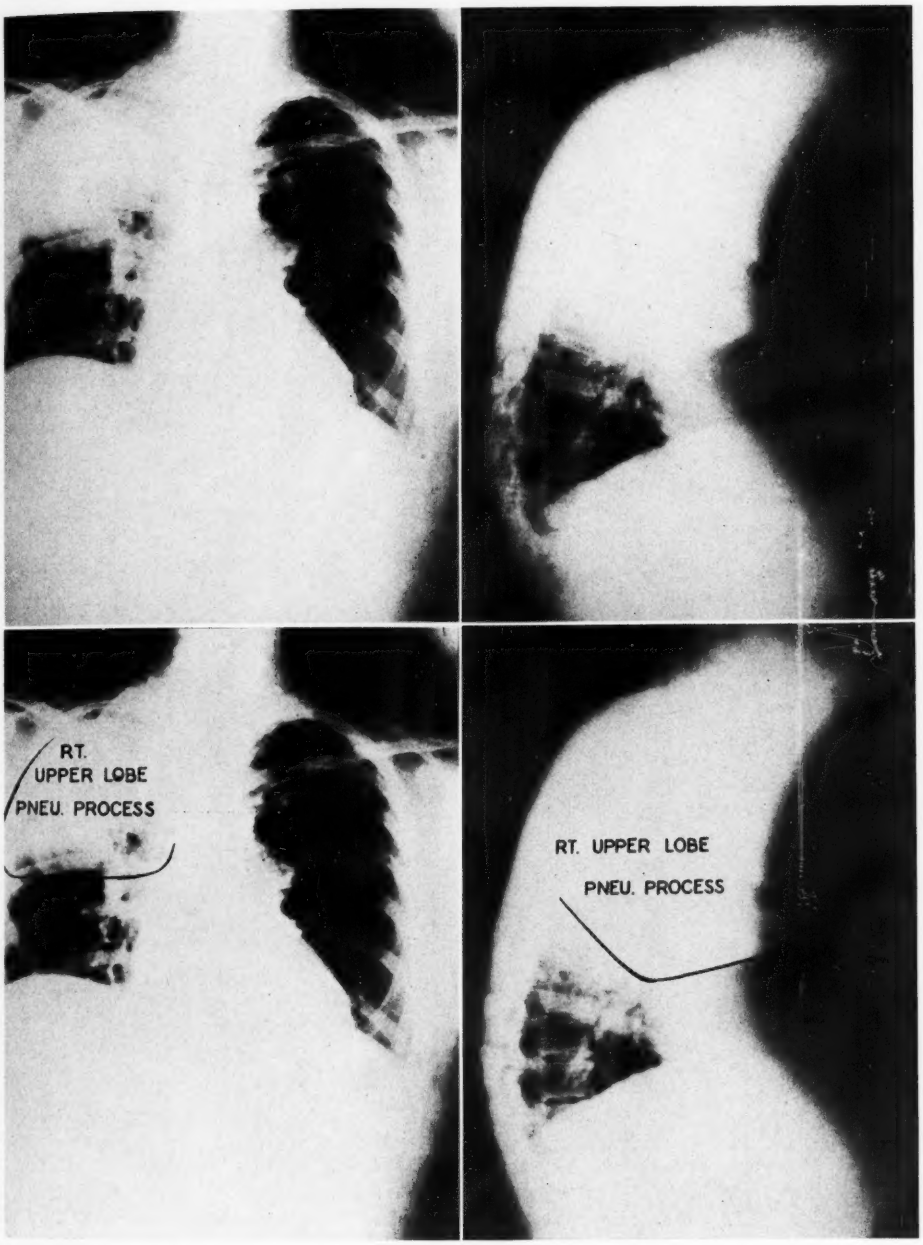


Fig. 1-*A*. Case 1. Pneumonic consolidation of the right upper lobe.

lobes may be affected. Notwithstanding such factors which may play a part in the distortion of these shadows, the general configuration of the affected parts of the lung closely simulate the diagrams experimentally produced.

In a previous article[3] one of us described

[3] RADIOLOGY, November, 1934, **23**, 629–634.

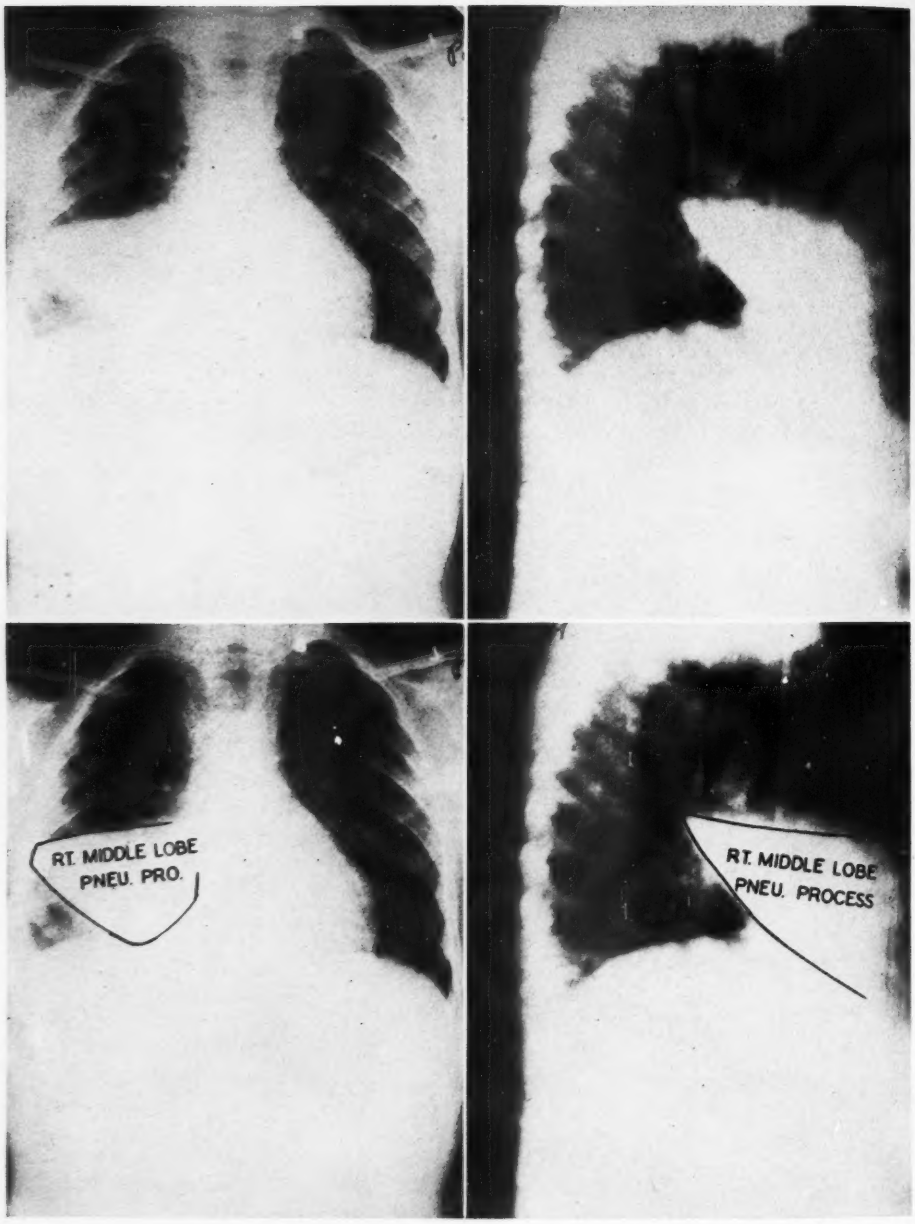
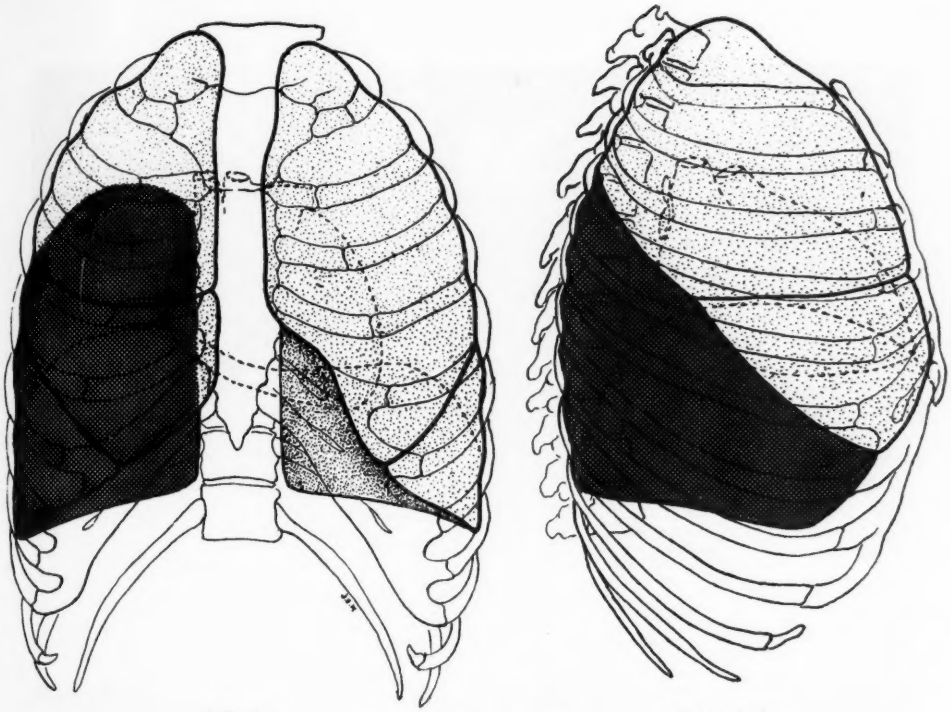


Fig. 2-*A*. Case 2. Pneumonic consolidation of the right middle lobe Note clearance at lateral margin of the base, the area occupied by the lower lobe. If the diaphragm is elevated, the shadow of the middle lobe is continuous with that of the liver.

the roentgenologic appearance of the potential interlobar spaces. The present study is a continuation of the subject, illustrating the affected spaces by clinical cases. In addition, we have included a study of the appearance of the various lobes of the lung, with illustrative cases.

Method.—Models of the lobes were made



A-P view

Lateral view

Fig. 3. Right lower lobe.

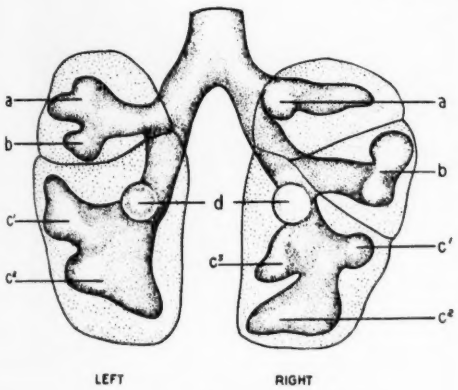


Fig. 4. Human embryo (11 mm.).

a, *b*, Anlage for bronchi to single left upper lobes.

c^1, c^2, Main bronchial anlage for left lower lobe.

a, Anlage of apical bronchus for right upper lobe.

b, Anlage of lateral first central bronchus for right middle lobe.

c^1, c^2, c^3, Main bronchial anlage for right lower lobe.

d, Separate origin of bronchial anlage for superior division of lower lobe.

From Keibel and Mall (*3*).

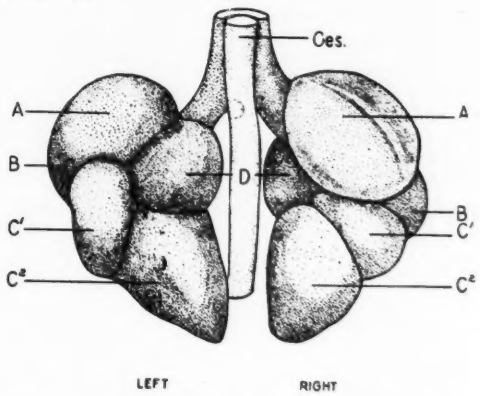


Fig. 5. Human embryo (13 mm.). Development of lung buds.

A, *B*, Lung buds for single left upper lobe.

C^1, C^2, Lung buds for inferior division of left lower lobe.

D, Lung bud for superior division of left lower lobe.

A, Lung bud for right upper lobe.

B, Lung bud for right middle lobe.

C^1, C^2, Lung buds for inferior division of right lower lobe.

D, Lung bud for superior division of right lower lobe. Failure of fusion of lung bud *D* with lung buds C^1, C^2 accounts for the presence of an anomalous fissure in the developed lung.

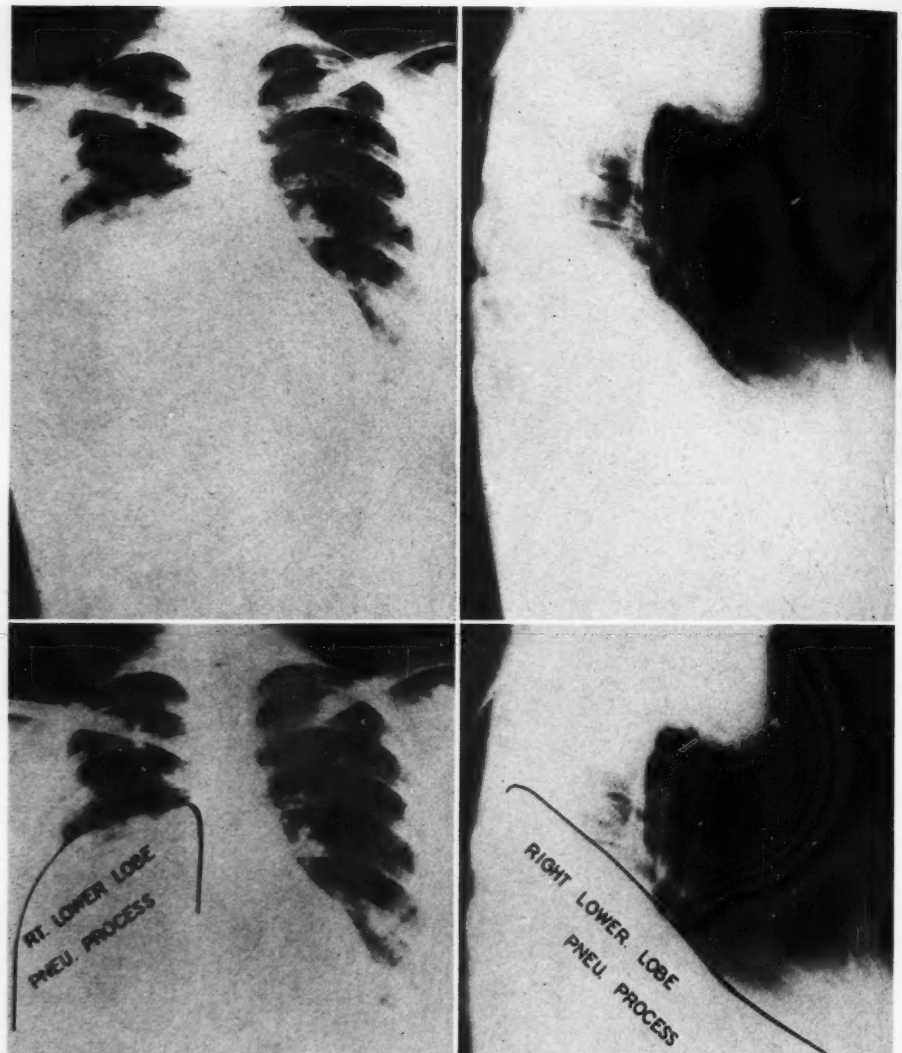
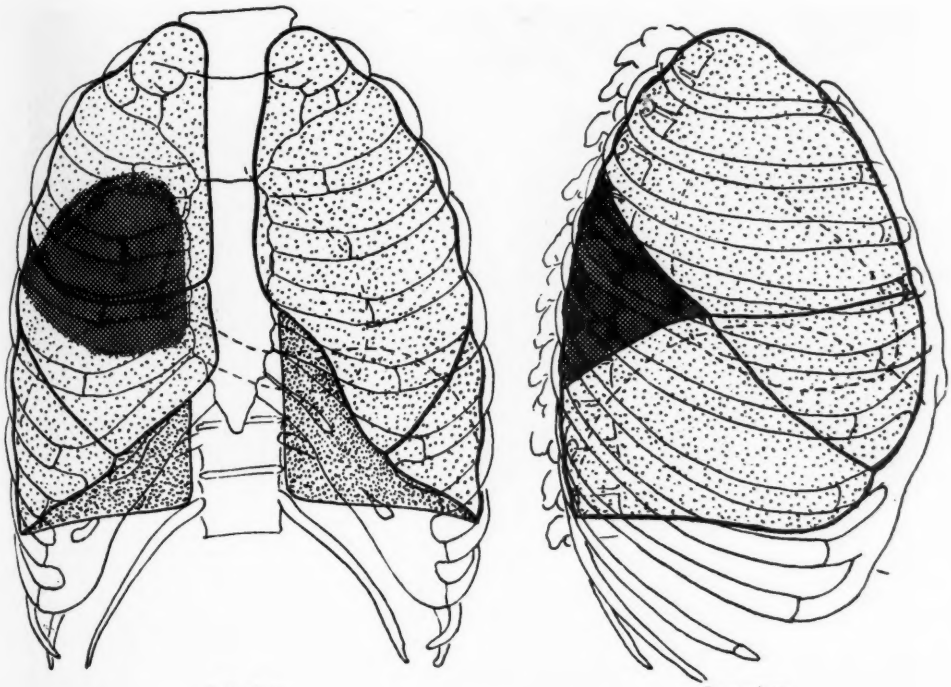


Fig. 3-*A*. Case 3. Lobar pneumonia of the right lower lobe. Confirmed at autopsy.

of paraffin, which is transparent to the x-ray. The models were fitted into a thoracic skeleton. To study the appearance and location of the individual lobe, lead foil about one-fourth millimeter in thickness was wrapped around the various lobes and films were taken in the postero-anterior and lateral positions. The ap-



A-P view Lat. view

Fig. 6. Superior division of right lower lobe.

pearance of each interlobar fissure was studied by placing the lead foil between the lobes and taking the films in the postero-anterior and lateral positions. Because of artefacts in the preparation of the skeleton, the roentgenograms obtained were subsequently diagrammed. In the prepared diagrams the position of the heart was indicated by dotted lines. The fissures of the surface of the lung as seen on the frontal and lateral surfaces were indicated by a heavy black line. The interlobar fissure studied was indicated by a heavy checkered shadow. A high diaphragm adds to the difficulty of interpretation. At full expiration the diaphragm rises to a height on a plane with the level of the lower border of the fourth rib, thus masking a considerable portion of the lung. The diaphragm was indicated on the diagrams at full expiration.

We have found these diagrams of great aid in the interpretation of many obscure shadows. At this point we wish to emphasize again the importance of a comparative study of the lateral and postero-anterior projections in arriving at conclusions as to the location of processes in the chest.

GENERAL DESCRIPTION OF THE LOBES AND INTERLOBAR FISSURES

The right lung is divided into three lobes—upper, middle, and lower—by two interlobar fissures; and the left lung is divided into two lobes—upper and lower—

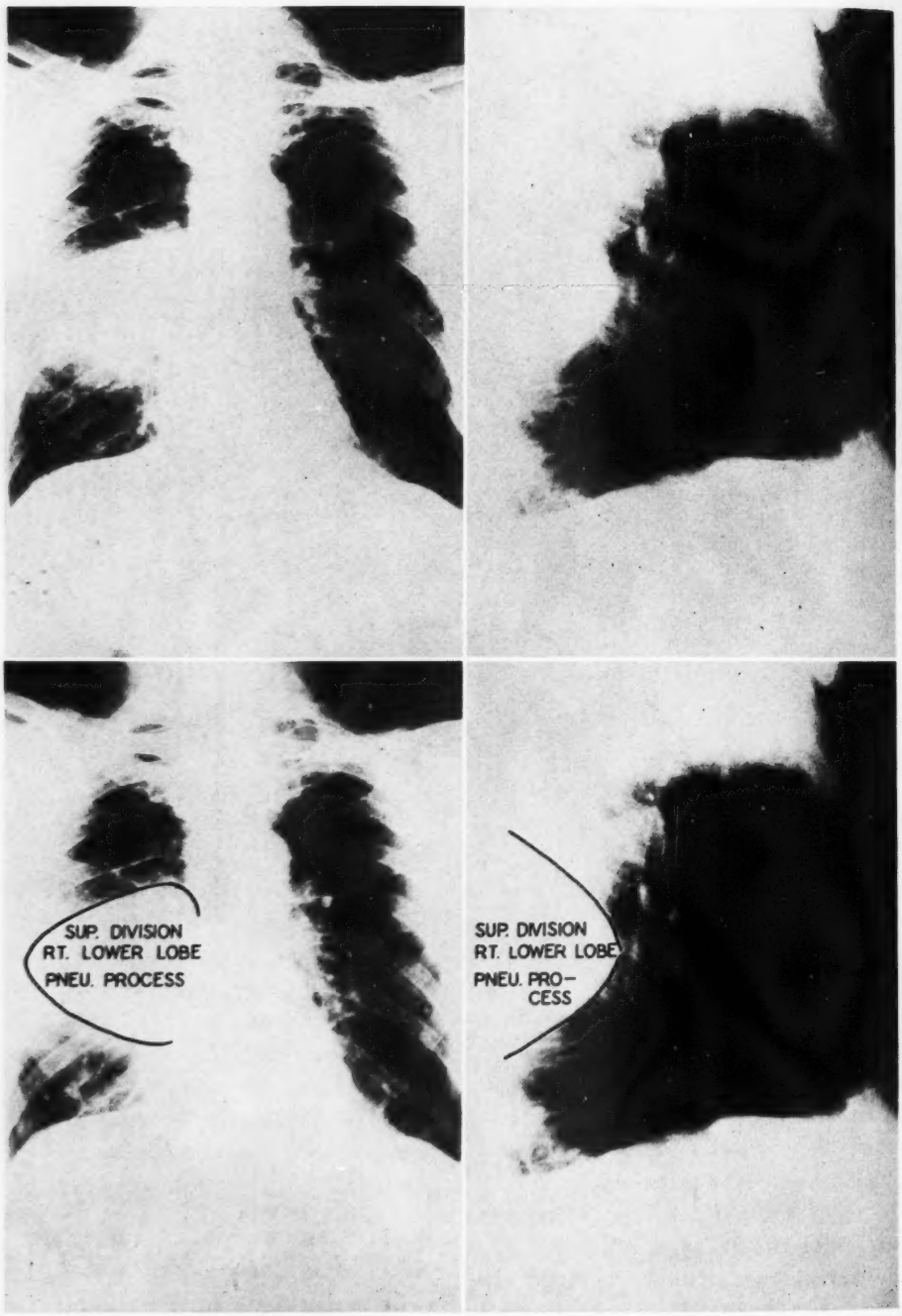
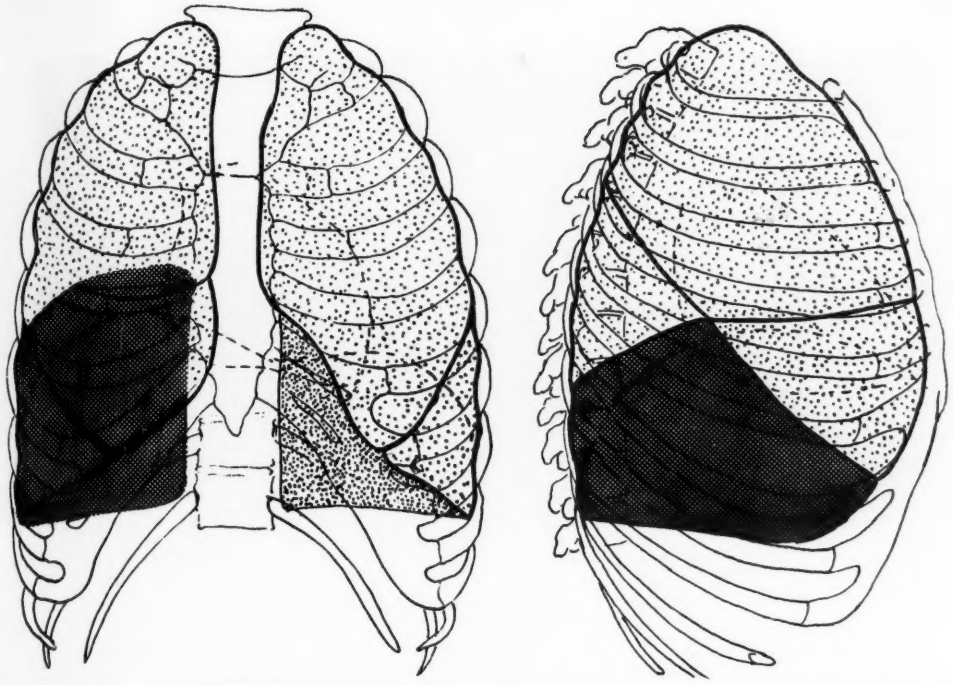
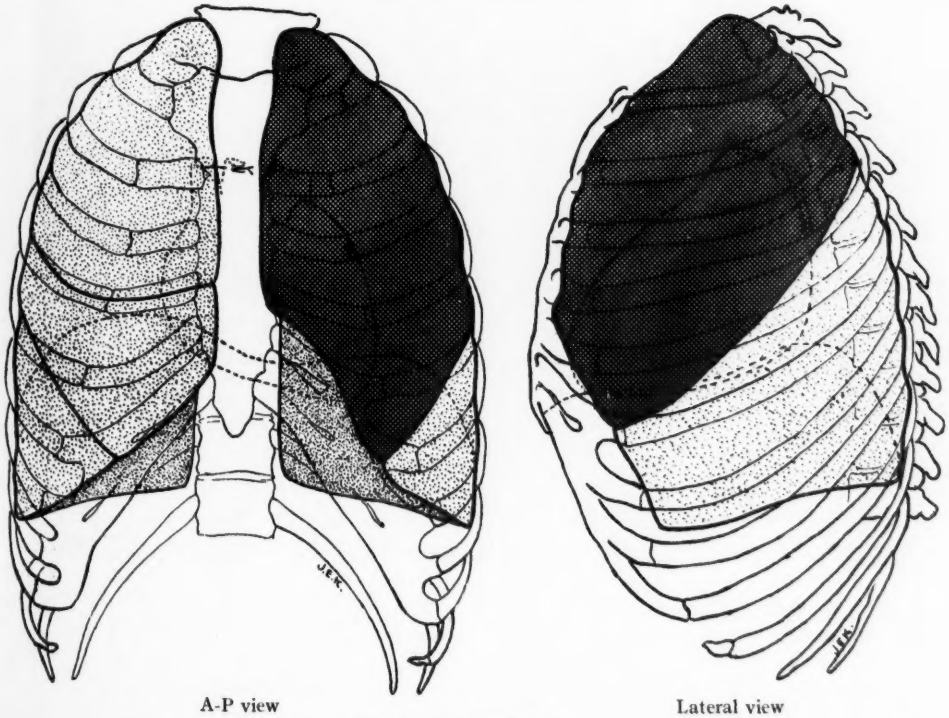


Fig. 6-*A*. Case 4. Pneumonic process superior division of the right lower lobe, which developed into a lung abscess. Note the posterior triangular appearance of this process in the lateral view.



A-P view Lat. view

Fig. 7. Inferior division of right lower lobe.



A-P view Lateral view

Fig. 8. Left upper lobe.

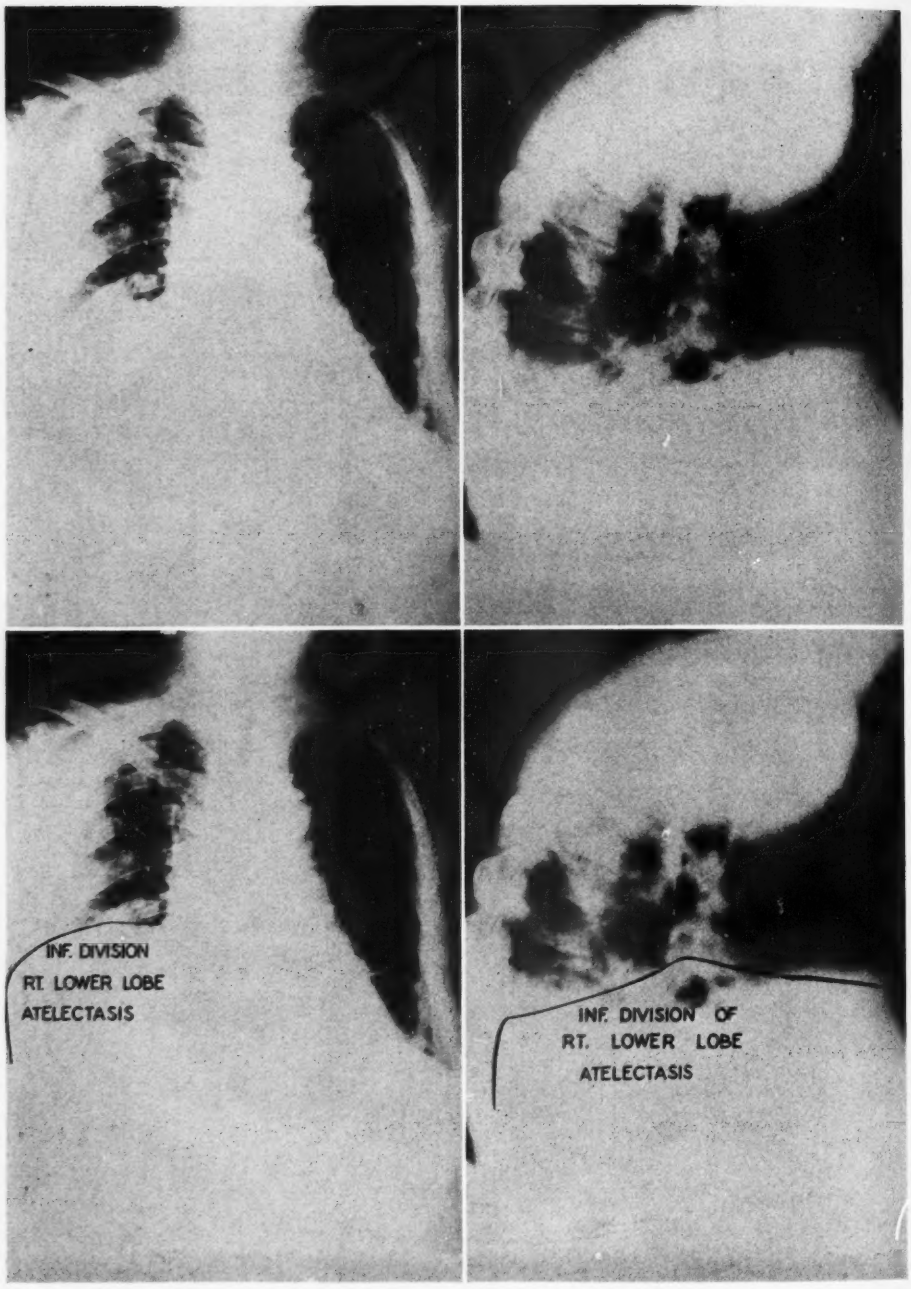
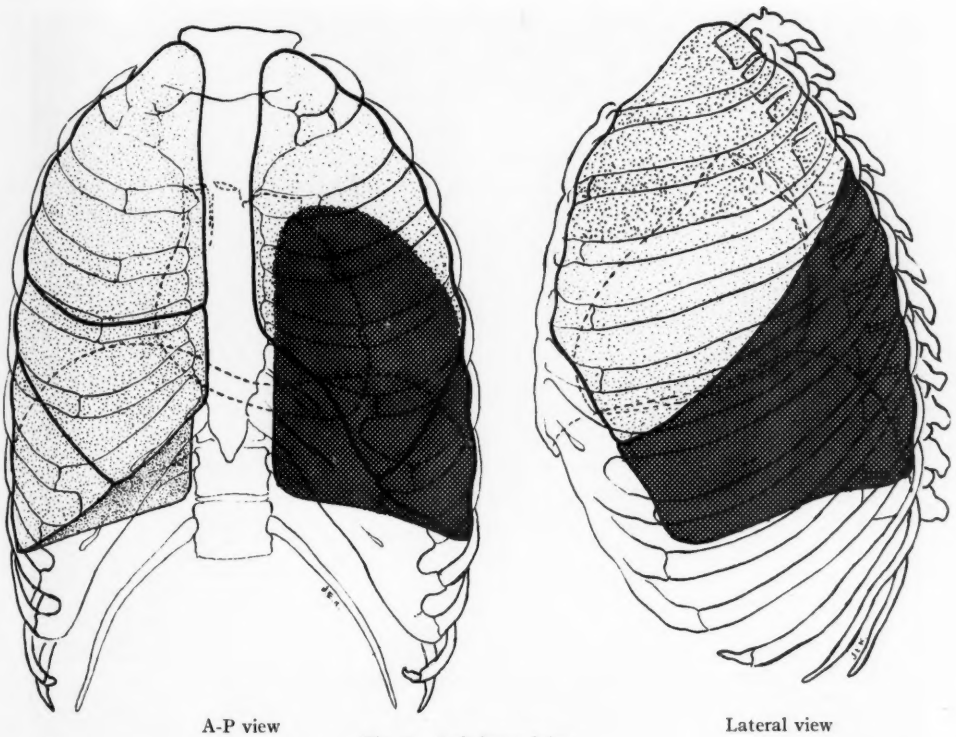


Fig. 7-*A*. Case 5. Atelectasis of inferior division of the right lower lobe as result of carcinoma of the bronchus. The shadow of this part of the lobe is continuous with the liver shadow and in the lateral view the posterior recess behind the diaphragm is obscured by this part of the involved lung.



A-P view Lateral view

Fig. 9. Left lower lobe.

by one interlobar fissure. The course of the major fissure on both sides is from the level of the spinous process of the third dorsal vertebra posteriorly, obliquely downward and forward to the costochondral articulation of the sixth rib anteriorly. It separates the superior lobe above and the inferior lobe below. On the right side another fissure extends horizontally forward from the axilla to the costochrondral articulation of the fourth rib. It divides the superior lobe into an upper and a middle lobe.

THE LOBES OF THE LUNG

Right Upper Lobe (Fig. 1).—The right upper is the superior lobe and on the roentgenogram occupies most of the chest in the postero-anterior view. The shadow of the diaphragm at full expiration extends almost to the shadow of the lower margin of this lobe. The lateral view shows the lobe filling the upper chest, corresponding closely to the rib cage. From the hilus the lower border extends forward horizontally to the anterior chest wall and backward obliquely to the posterior chest wall. The upper lobe overlaps the superior part of the lower lobe in the postero-anterior view.

Right Middle Lobe (Fig. 2).—The right middle lobe is an anterior lobe, triangular in shape, with the apex at the hilus. In the postero-anterior view it occupies the lower half of the chest. The upper border is horizontal, extending outward from the hilus. The lateral margin curves medially so that a part of the lower lobe is visible at the base in the axilla. However, this part of the lobe is customarily not seen; it is usually obscured by the diaphragm. The lateral view clearly demonstrates the triangular and anterior position of this lobe. In this position, the shadow of the lobe superimposes the cardiac shadow, from which it must be differentiated.

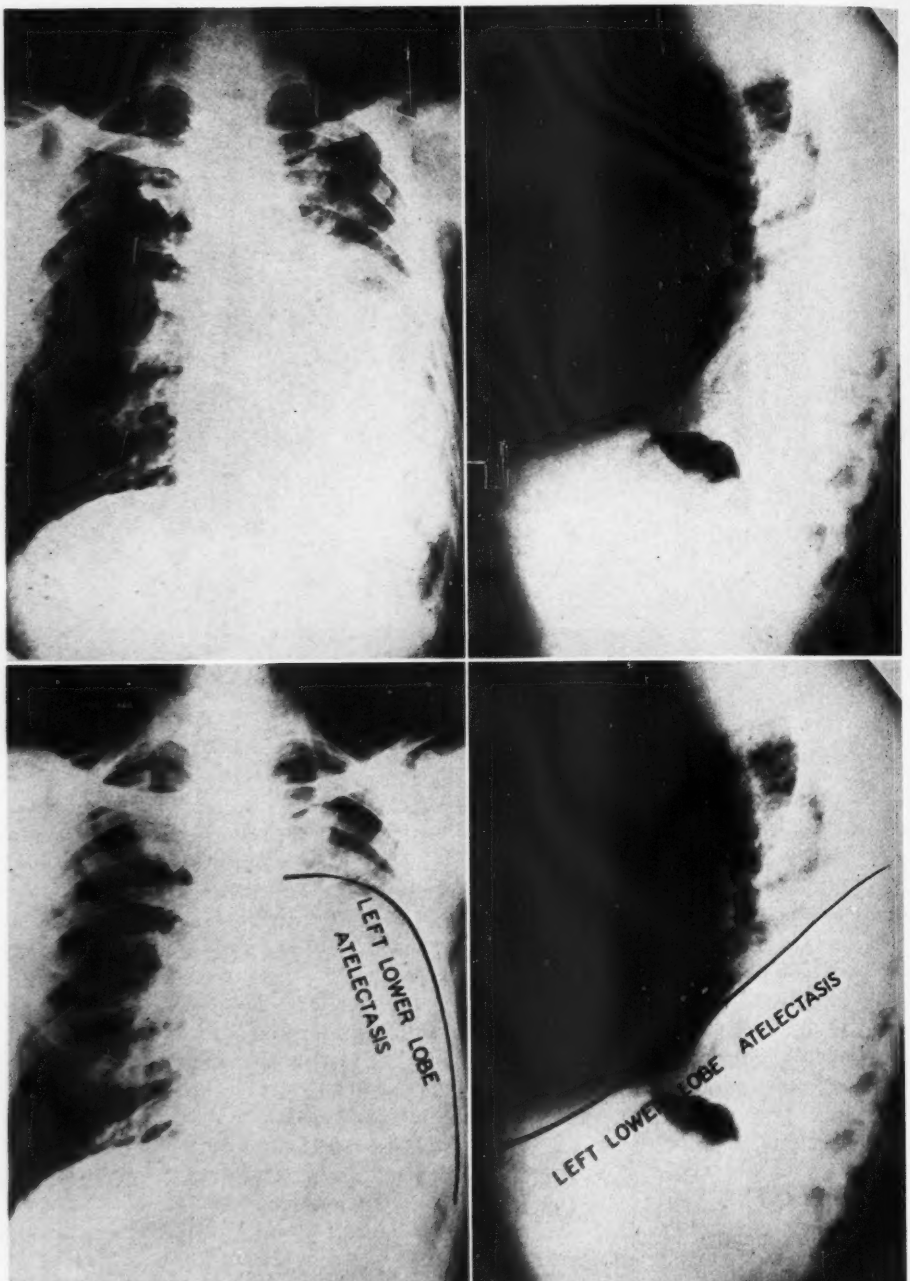
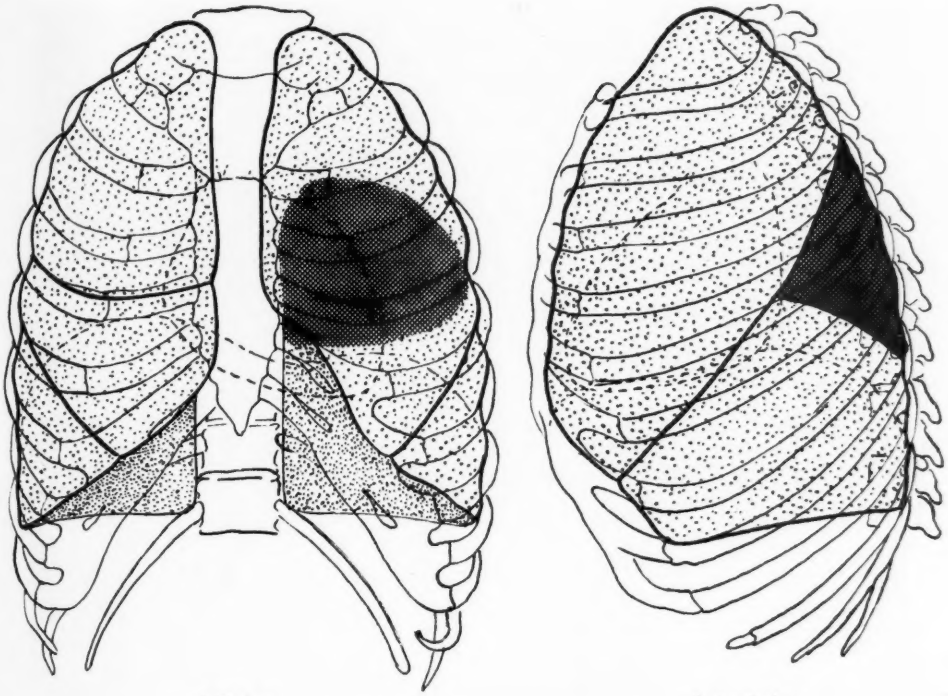


Fig. 9-A. Case 6. Atelectasis of the left lower lobe as result of primary carcinoma of the main bronchus of the left lower lobe.



A-P view Lateral view

Fig. 10. Superior division of left lower lobe.

The Right Lower Lobe (Fig. 3).—The right lower lobe occupies the posterior and inferior part of the chest. It extends as high as the fourth rib posteriorly, and in the postero-anterior view is projected on a level with the second rib anteriorly. It is found in the axilla and for a small area on the anterior chest wall, where it borders the middle lobe.

In a previous article[3] one of the authors described the lower lobes on either side as divided into two parts, a superior and an inferior division. The division of this lobe has an embryological origin. There is a distinct bronchial distribution to each of its parts and a fissure separating them may be present. Figure 4 demonstrates the separate origin of the bronchial anlage. Figure 5 explains the formation of the fissures between the lobes. Failure of fusion between the separate buds of the lower lobes accounts for the anomalous fissures often present.

Superior Division (Fig. 6).—The superior division of the lower lobe is the upper posterior part of the lobe, well seen in the lateral view, occupying a triangular area, with the apex at the hilus. It is that part of the lower lobe which for the most part lies behind the superior lobe. When the diaphragm is low, it appears to occupy the central part of the chest in the postero-anterior view. With an elevated diaphragm its shadow may be continuous with the liver shadow. The lateral view clearly establishes the triangular posterior position. Lung abscesses are most frequently found in this part of the lung. Primary carcinoma of the lung is infrequently found here. Pneumonia of the lower lobe also is more frequent in this part of the lobe, and because of its central appearance on the roentgenogram, is often called "central pneumonia." The physical signs of this process are found in a small circumscribed area posteriorly between the scapula and

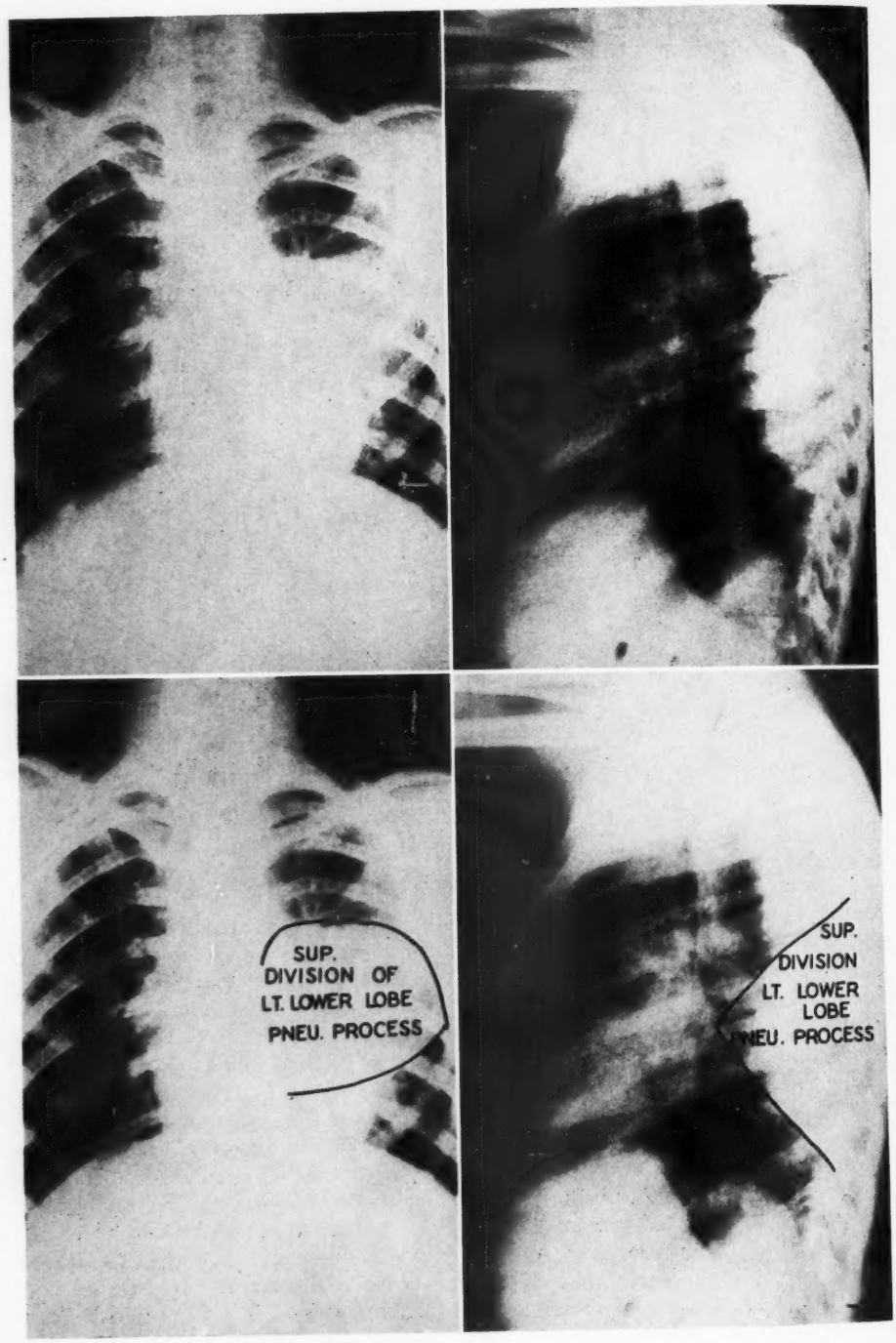
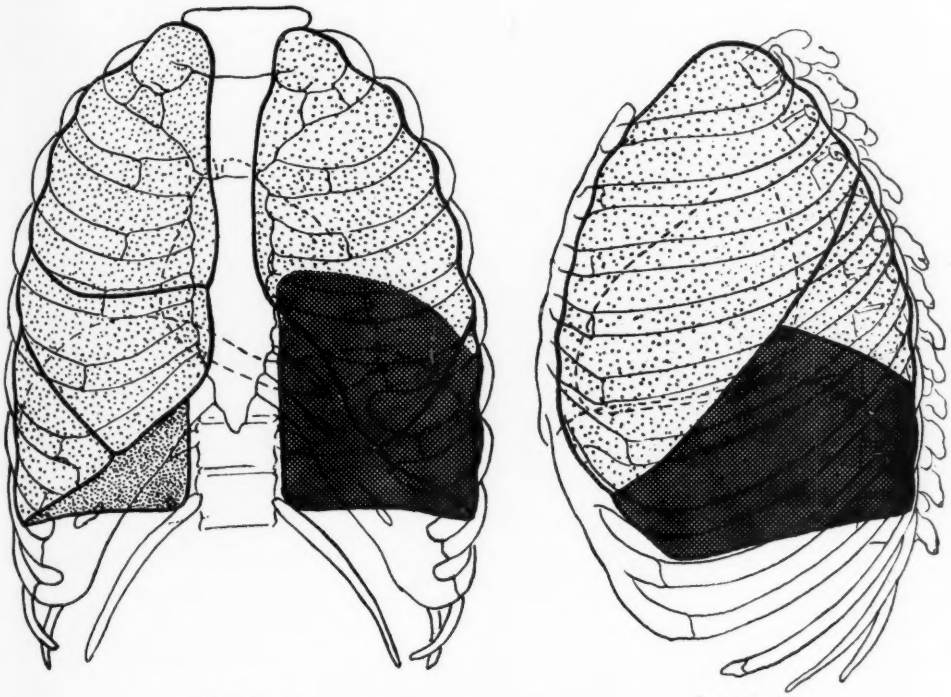


Fig. 10-*A*. Case 7. Pneumonia involving the superior division of the left lower lobe.



A-P view Lateral view

Fig. 11. Inferior division of left lower lobe.

spine and between the fourth to eighth ribs.

Inferior Division of the Lower Lobe (Fig. 7).—The inferior division is the larger part of the lower lobe. It extends posteriorly, anteriorly, and down to the diaphragm. Its shadow is continuous with the shadow of the liver. In the postero-anterior view the upper border may be horizontal or oblique, depending on the nature of the disease process, *i.e.*, whether consolidated or atelectatic. The upper border in the lateral view is usually tented, although it too may be horizontal, again depending on the nature of the disease process. Atelectasis is frequently found here, especially post-operative. A study of the diagram explains why physical signs of atelectasis of this part of the lung are frequently absent and considerable lung involvement is seen on the roentgenogram. If the atelectasis is of long duration, there is considerable diminution in size of the involved lung. The superior division of the lower lobe will become emphysematous and fill the posterior recess behind the diaphragm. Normal breath sounds and resonance will be found posteriorly. Likewise, anteriorly, the clear middle lobe will mask the involved lung. There may be a few signs in the axilla. The dullness, if found, is continuous with the liver dullness.

The Left Lung.—The left lung does not present as many complicating factors in its appearance on the roentgenogram as does the right lung. The presence of only two lobes, with the one interlobar fissure, reduces the number of possible combinations of lobes involved and interlobar effusions with its superimposed shadows.

It is interesting to note the infrequency of involvement of the entire left upper lobe by pneumonic consolidation and the infrequency of effusion in the interlobar fissure. Of a total of 370 cases of pneumonia reviewed at Mt. Zion Hospital, only one

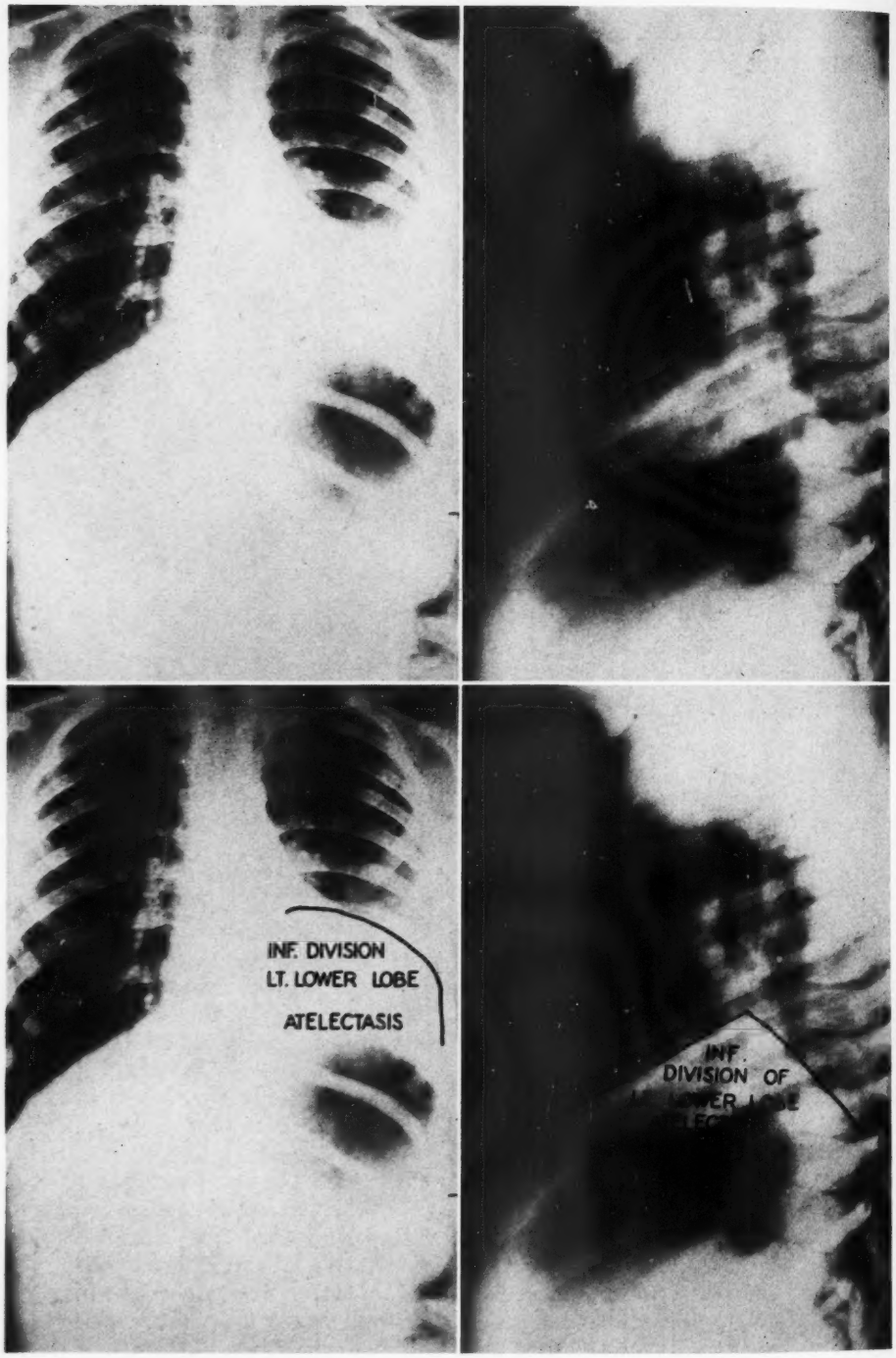


Fig. 11-*A*. Case 8. Atelectasis of inferior division of the left lower lobe as the result of a mucous plug. The lateral view demonstrates the tented appearance of this part of the lobe. The normal lung overlies this process both anteriorly and posteriorly which accounts for the fact that physical examination may not reveal the underlying process.

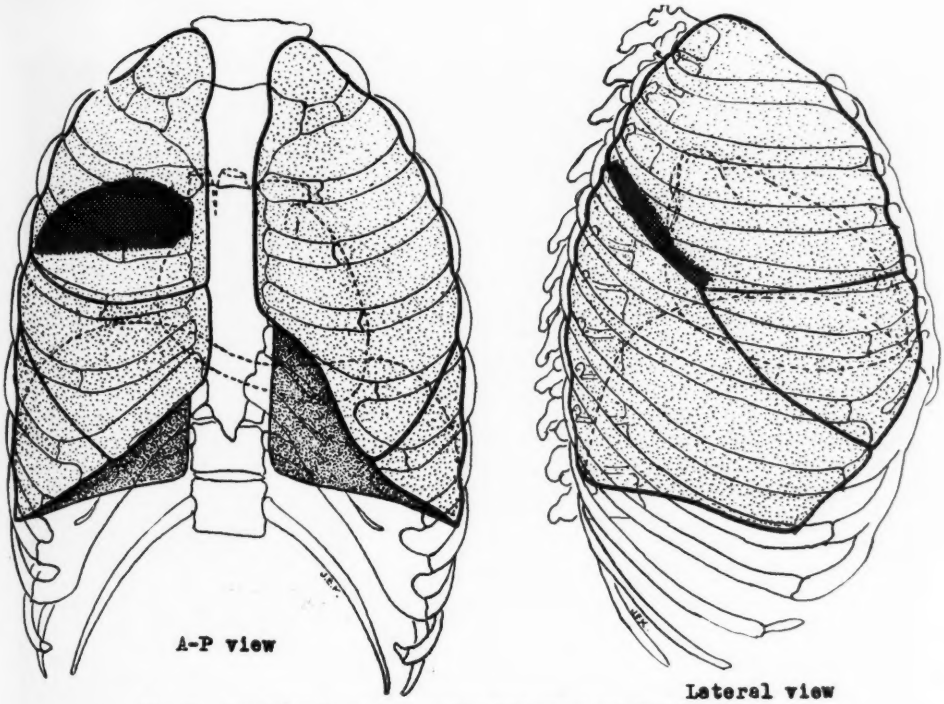


Fig. 12. Interlobar fissure between the right upper and right lower lobes.

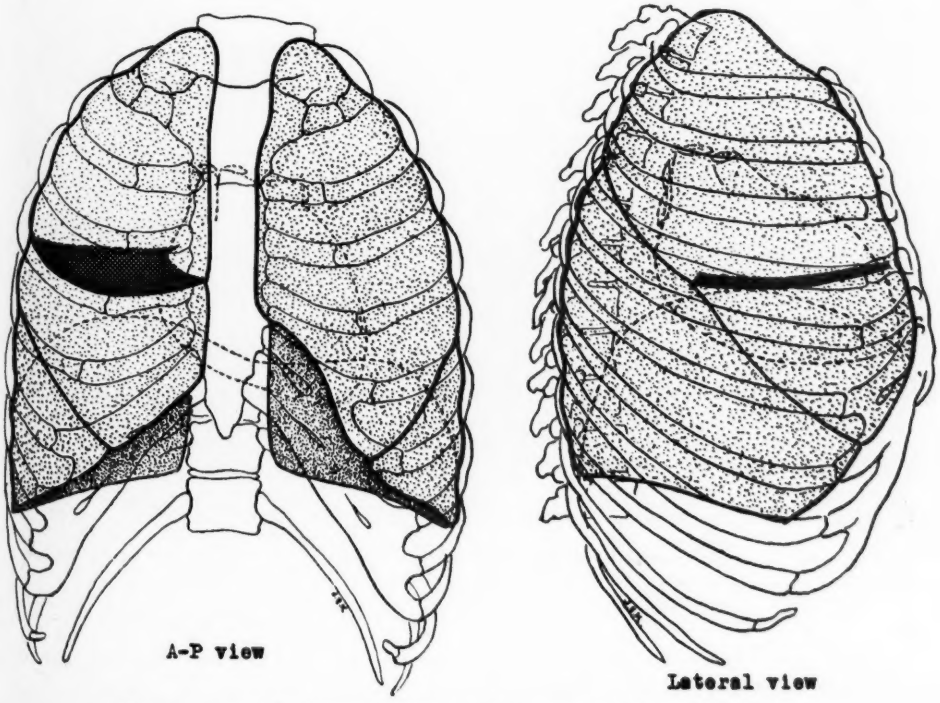


Fig. 13. Interlobar fissure between the right upper and right middle lobes.

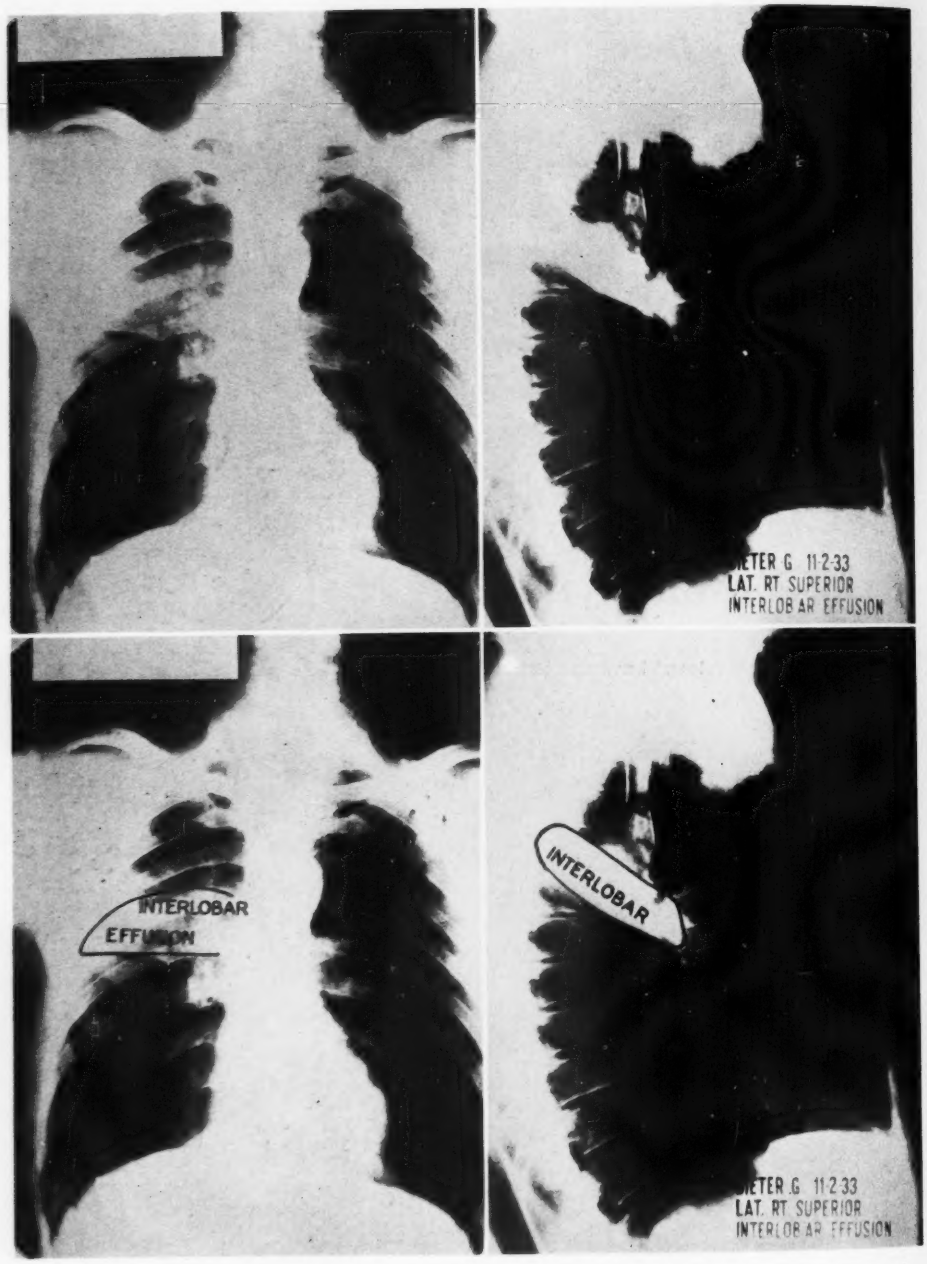


Fig. 12-*A* Case 9. Interlobar effusion between the right upper and right lower lobes. This was a neighboring effusion to a primary lung carcinoma of the right upper lobe

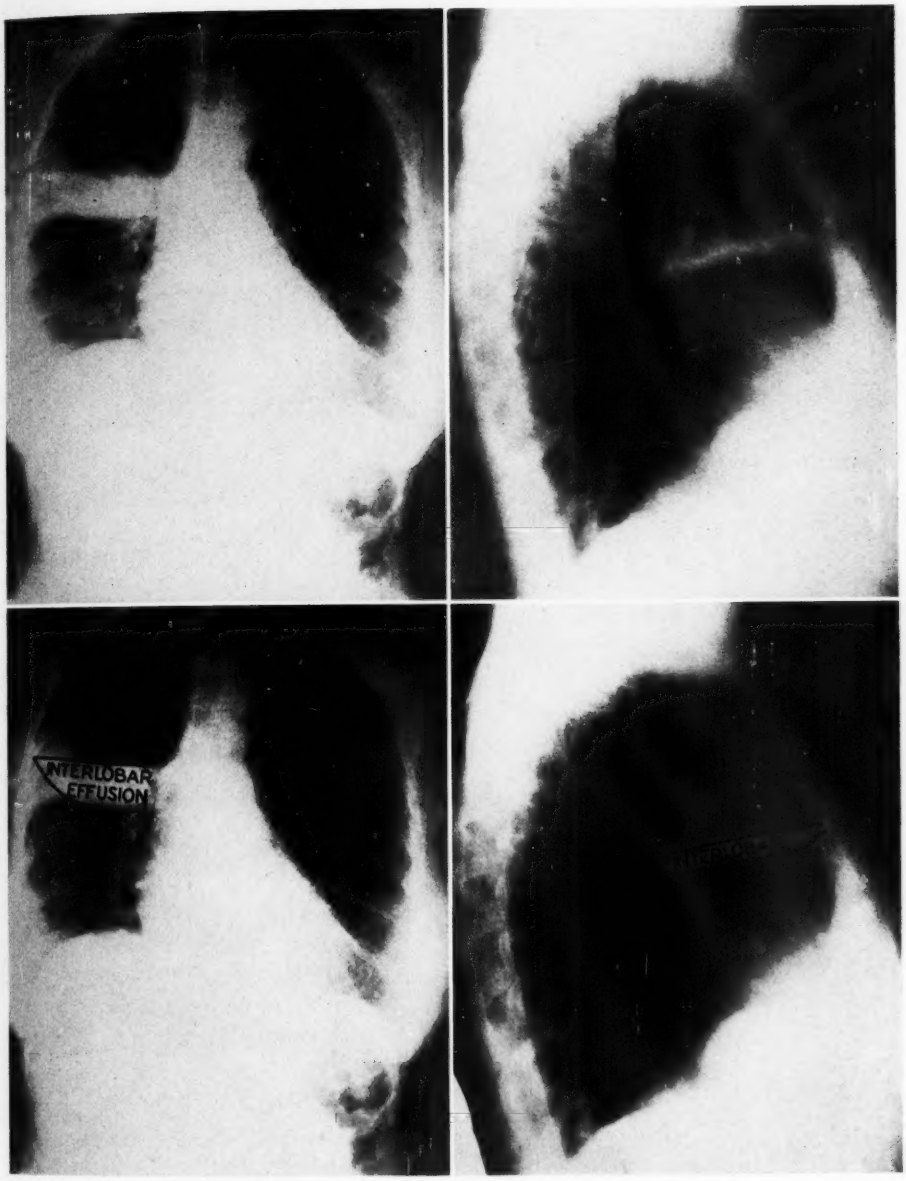


Fig. 13-*A*. Case 10. Interlobar effusion between the right upper and right middle lobes. This effusion followed a pneumonia in the right upper lobe.

case of pneumonia was found to be limited to the left upper lobe. This was a diffuse bronchopneumonia which later spread to the other lobes. One hundred cases of interlobar effusions were reviewed—of this number only two were found on the left side and both were incomplete effusion, *i.e.*, not filling the entire interlobar space. The left lower lobe was found to be as frequently involved by pneumonia as the right lower lobe. It also showed the similar division into a superior and inferior part.

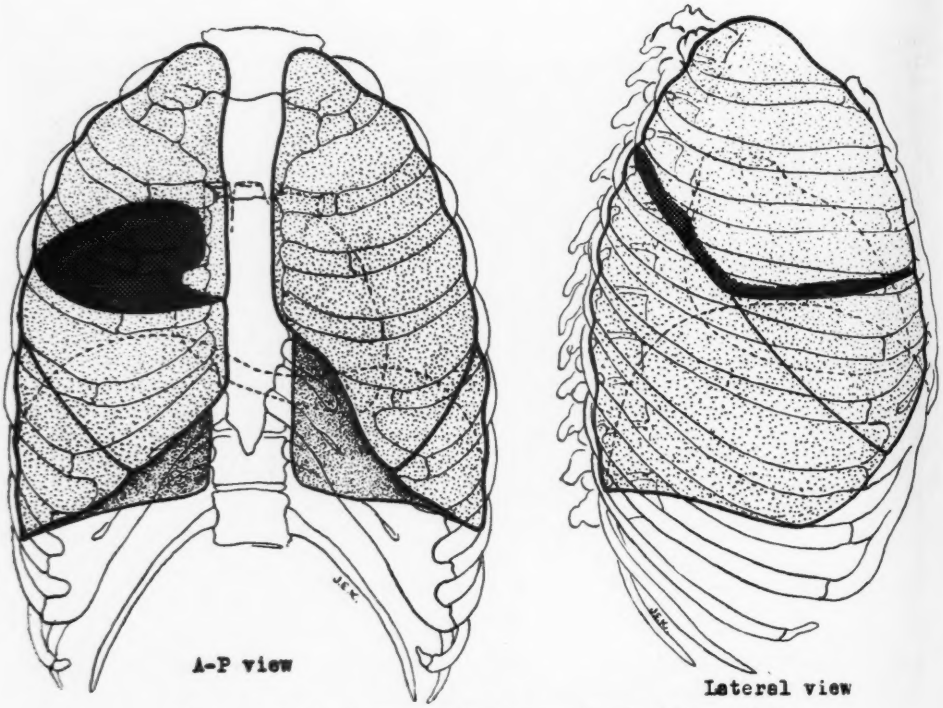


Fig. 14. Interlobar fissure completely surrounding the right upper lobe.

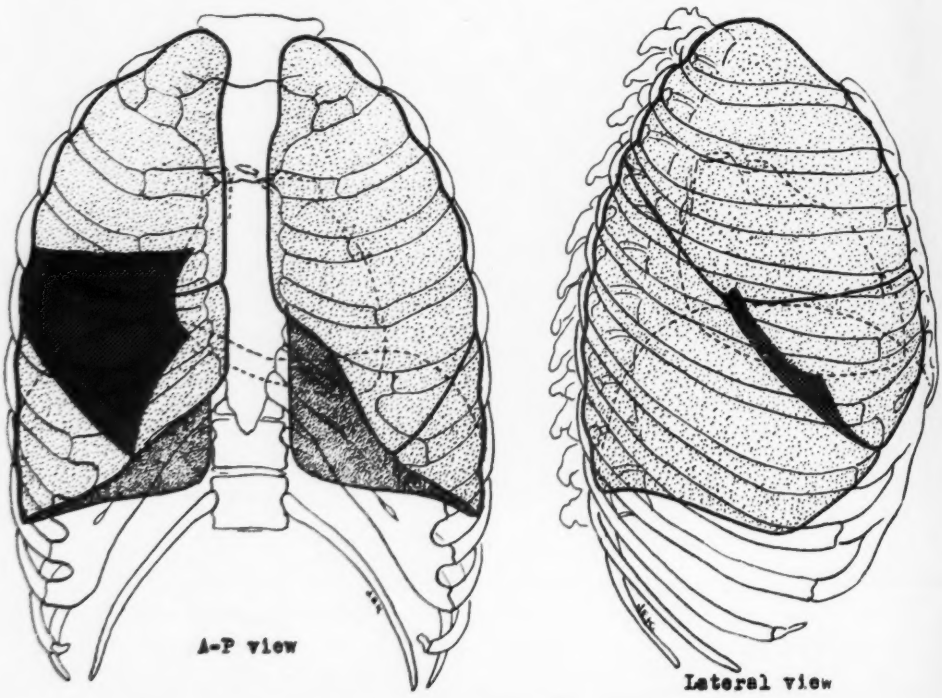


Fig. 15. Interlobar fissure between the right middle and right lower lobes.

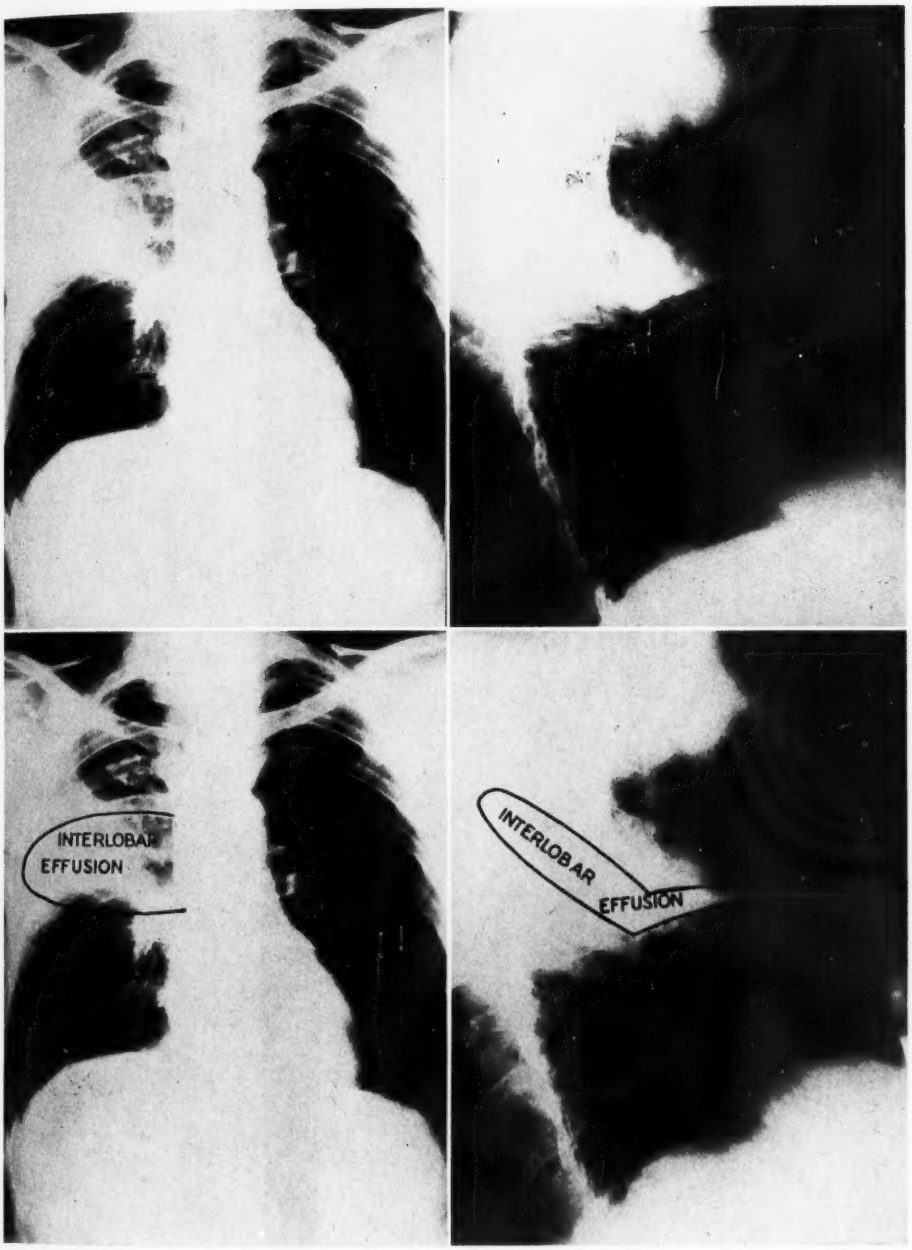


Fig. 14-*A*. Case 11. Interlobar effusion between the right upper and lower lobes posteriorly, and the right upper and middle lobes anteriorly. This effusion followed a pneumonia in the right upper lobe. Involvement of both these fissures results in a centrally located area of lessened density which may be mistaken for the breaking down of the lung parenchyma into an abscess cavity. The course of this patient was not that of a lung abscess. The process promptly cleared.

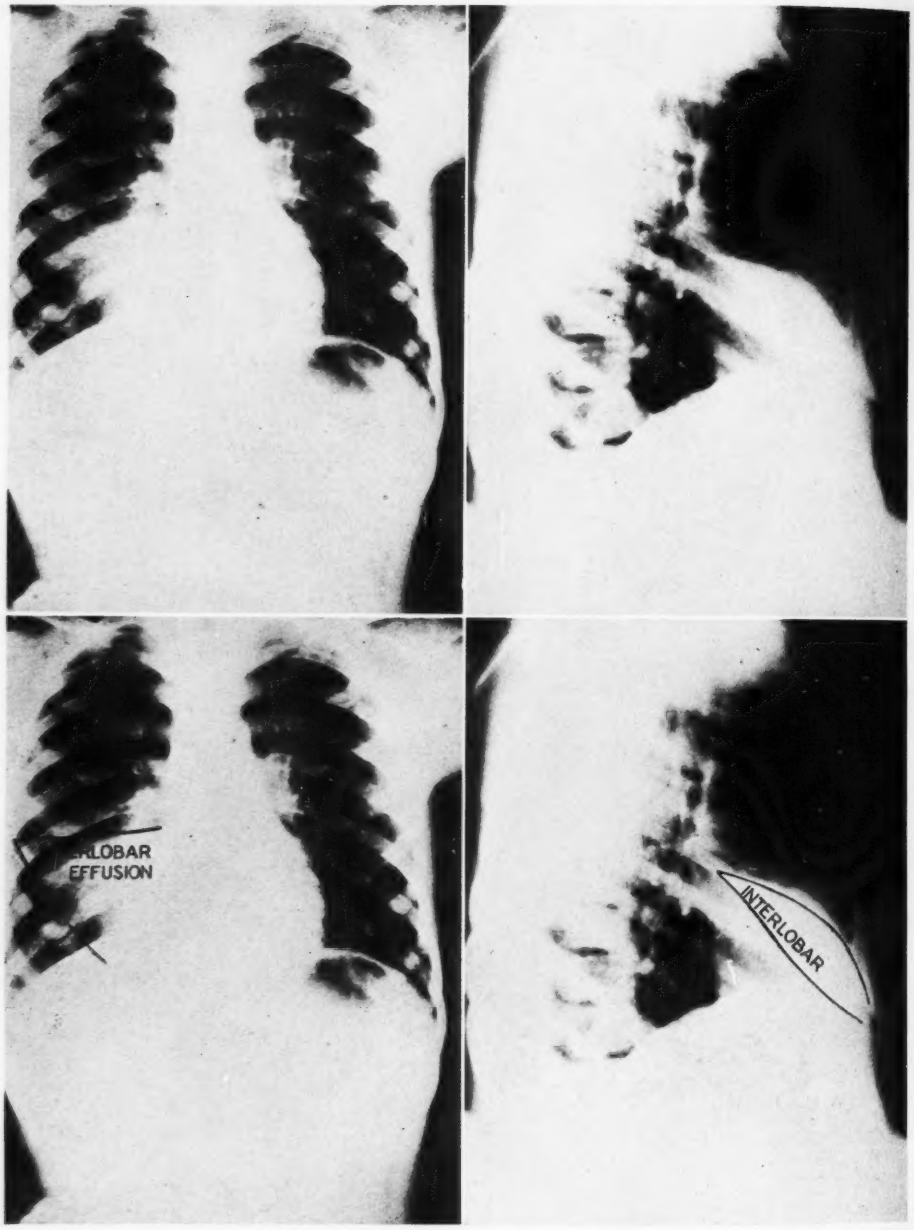


Fig. 15-*A*. Case 12. Interlobar effusion between the right middle and right lower lobes. This effusion often extends laterally so as to occupy the entire interlobar fissure. If the entire fissure is involved, the shadow in the anteroposterior view would be similar to that in Figure 2-*A*. The lateral view demonstrates the oblique position of this shadow with the tapering at both ends. This view is essential to establish the diagnosis, and differentiates a consolidated middle lobe from an interlobar effusion between the middle and lower lobes.

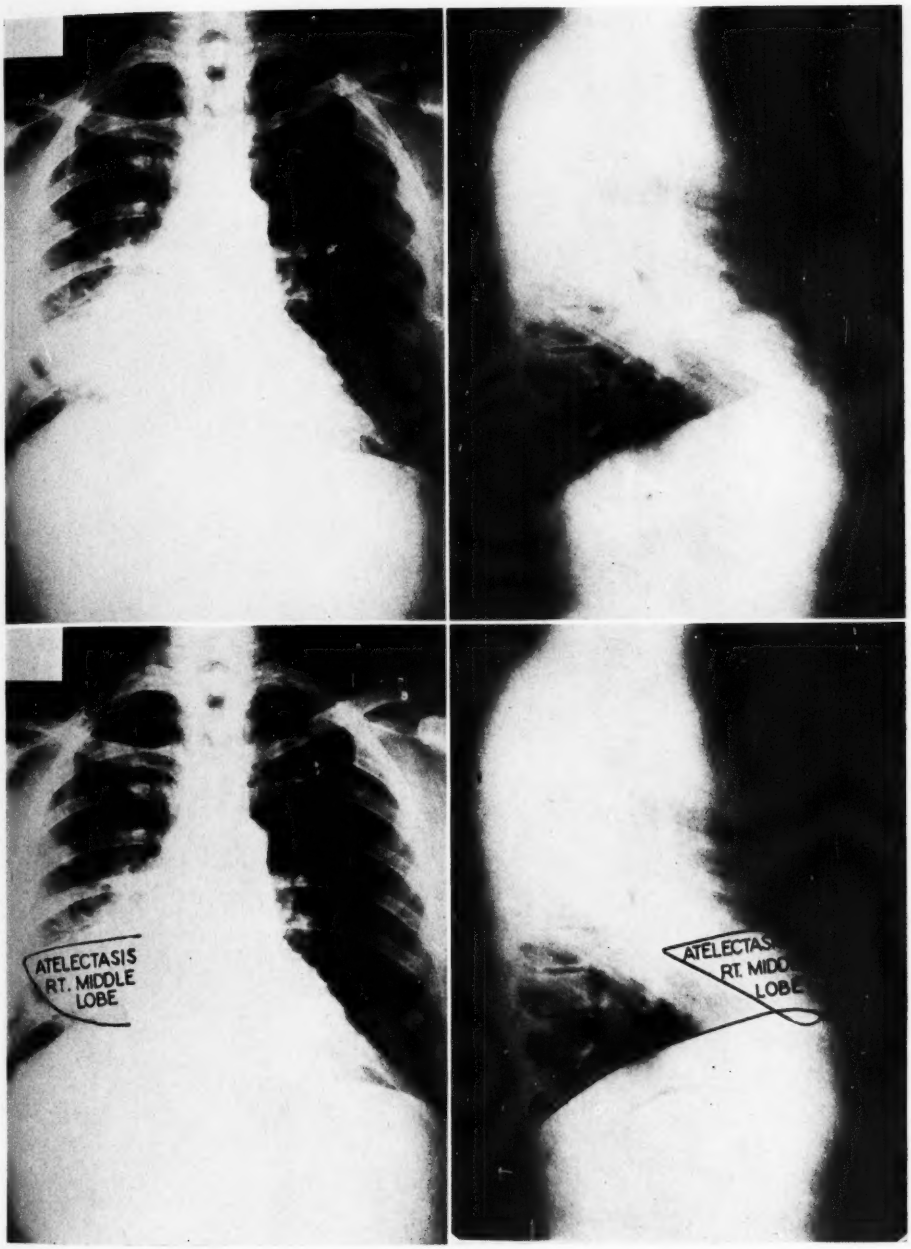


Fig. 16. Case 13. Atelectasis of the right middle lobe as a result of a primary bronchial carcinoma. Note the small size of the middle lobe. The lateral view may be confused with an interlobar effusion. The anterior tip of this shadow (in the lateral view) is not tapered as is seen in the interlobar effusion (Figure 15-*A*). The base is broad as is seen in the consolidated middle lobe (Figure 2-*A*).

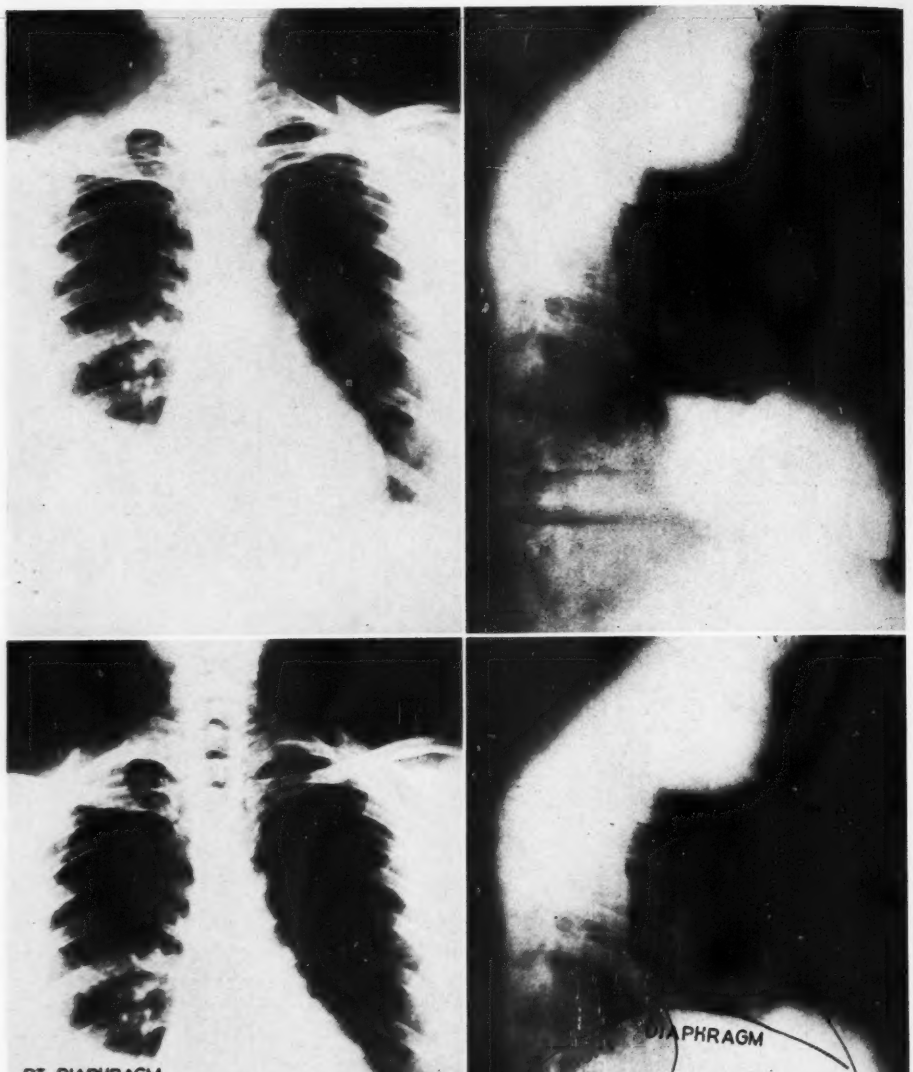


Fig. 17. Case 14. Superimposed cardiac shadow and elevated diaphragm. In the lateral view is seen an oblique dense shadow, tapering toward the tips, extending from the hilus forward and downward. This superimposed shadow has often been misinterpreted as interlobar effusion. Comparing the anteroposterior view to that of the middle lobe consolidation (Figure 2) and the fissures between the middle and lower lobes (Figure 15), it is noted that the broad shadow extending from the hilus toward the axilla and base is absent and the right diaphragm is elevated.

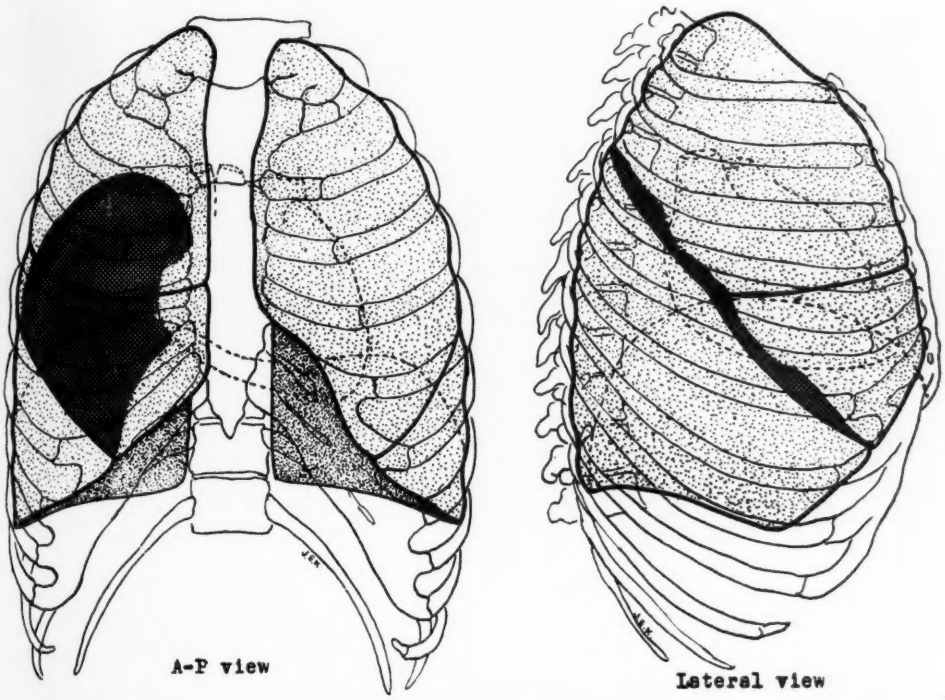


Fig. 18. Interlobar fissure bordering the upper surface of the right lower lobe (the major fissure separating the lobes).

The Left Upper Lobe (Fig. 8).—The shadow of this lobe on the roentgenogram is a shadow similar to the combination of the right upper and middle lobes. It occupies the upper and anterior part of the chest and overlies most of the cardiac shadow. No cases of consolidation limited to this lobe were found suitable for reproduction in the series studied.

The Left Lower Lobe (Fig. 9).—The left lower lobe is similar in appearance, size, and position to the right lower lobe. It is similarly divided into a superior and inferior division.

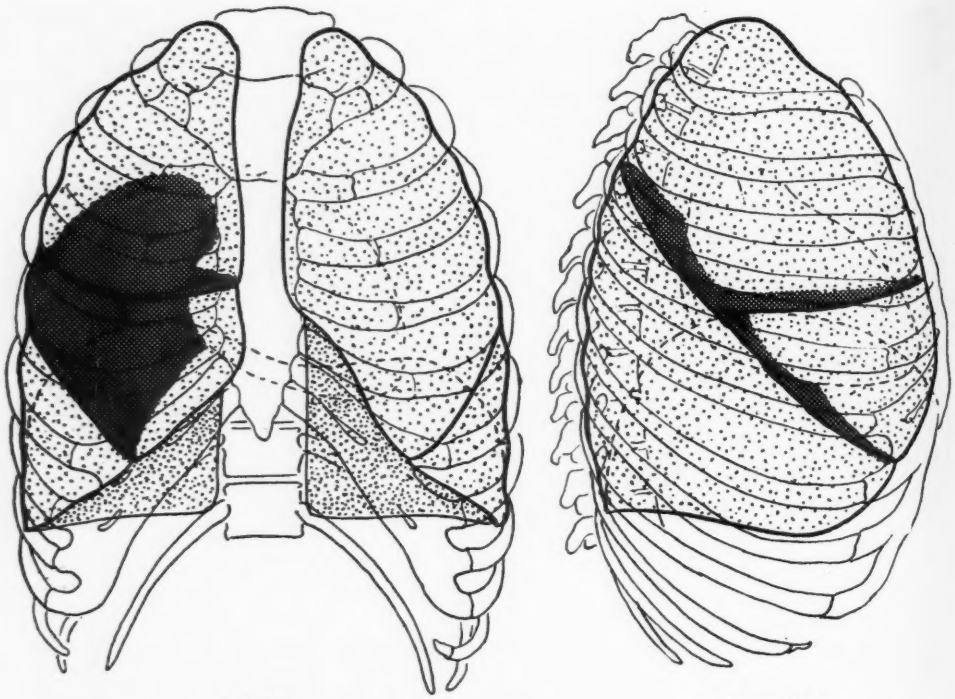
Superior Division of Left Lower Lobe (Fig. 10).—The appearance of this part of the lobe is similar to the superior division of the right lower lobe.

Inferior Division of Left Lower Lobe (Fig. 11).—This part of the lung is also similar to the counterpart on the right side.

THE INTERLOBAR FISSURES

Interlobar Fissure between the Right Upper and Right Lower Lobes (Fig. 12).—This part of the chest is the only place where the right upper and lower lobes are to contact. This fissure extends from the hilus obliquely upward. In the postero-anterior projection this fissure appears as a broad shadow which is often misinterpreted as lung involvement. The lower border appears as a horizontal line extending outward from the hilus. The upper border is convex, corresponding to the contour of the upper border of the lower lobe. The lateral view clearly demonstrates the oblique position of this fissure.

Physical signs of involvement of this fissure by an effusion may be absent. This is easy to understand because this fissure approaches the chest wall only in the axilla —its narrowest part. In front and in back



A-P view Lateral view

Fig. 19. Interlobar fissure between all the lobes on the right side.

of this fissure is lung parenchyma which, if not involved, would not transmit the dullness of an effusion.

Interlobar Fissure between Right Upper and Right Middle Lobes (Fig. 13).—This fissure extends forward from the hilus to the anterior chest wall. On the postero-anterior projection the lower border is convex and corresponds to the shape of the lower margin of the upper lobe. The upper border is horizontal, extending from the hilus to the axilla. The lateral view shows the fissure to extend forward almost horizontally, from the hilus to the anterior chest wall.

A study of the diagram illustrates why physical signs of an effusion in this fissure would be absent. Again we have the process overlapped by considerable lung which, if not involved, would not transmit the dullness of the effusion.

The Fissure Completely Surrounding the Right Upper Lobe (Fig. 14).—The shadow cast by this fissure is a combination of the shadows obtained in Figures 12 and 13. The upper border is convex, corresponding to the contour of the upper margin of the lower lobe. The lower border has the shape of the lower border of the upper lobe on the anterior chest wall. Because of the broad shadow obtained in the postero-anterior view, which is less dense centrally near the hilus, involvement of these two fissures has often been misinterpreted as lung involvement and frequently as a lung abscess. Since interlobar effusions most frequently follow pneumonia and account for the subsequent temperature, and lung abscesses are often complications of pneumonia, the interpretation of this shadow as a lung abscess is a pitfall which must be guarded against. The lateral view clearly demonstrates the contour of this fissure. It is the posterior fissure, lying obliquely, which accounts for the broad shadow.

Interlobar Effusion between the Right

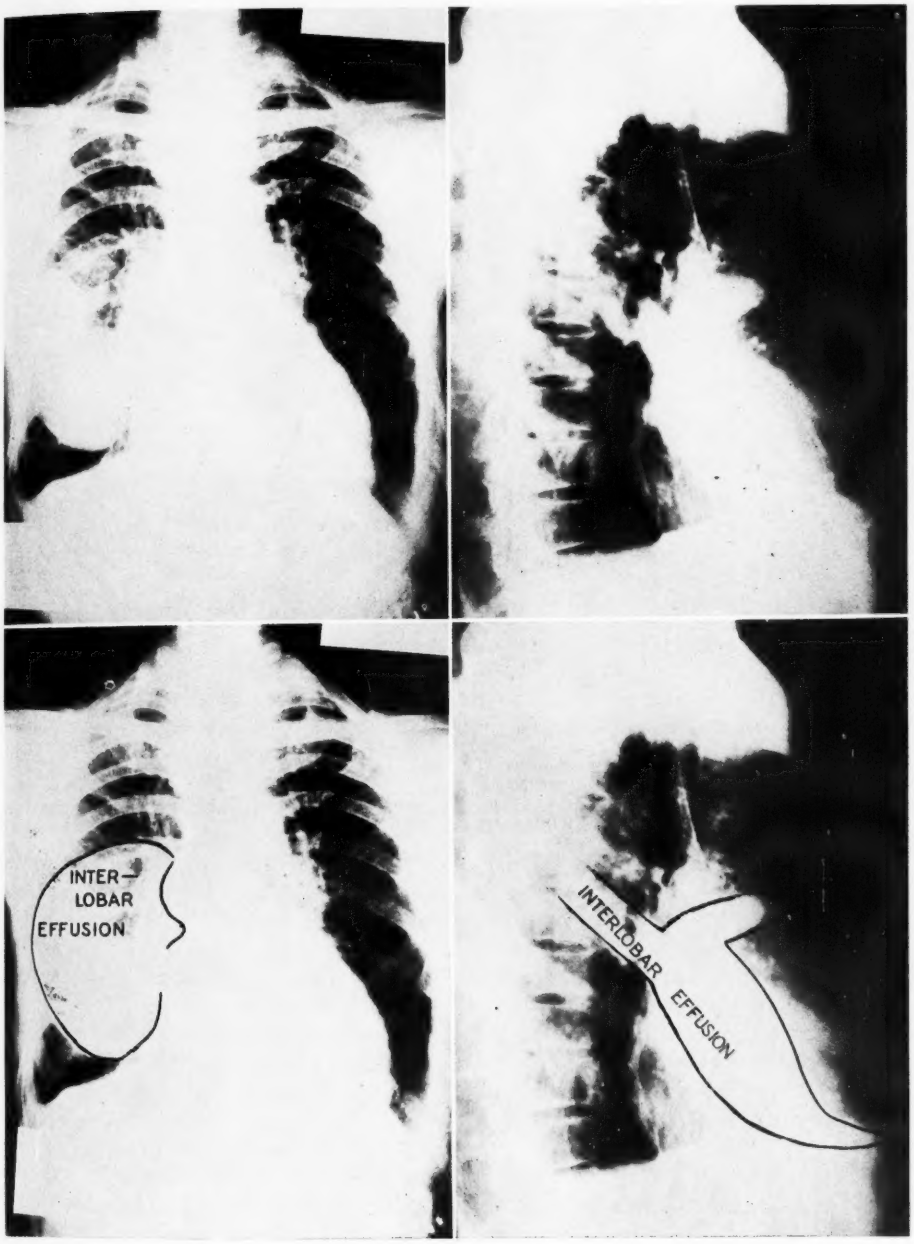


Fig. 19-*A*. Case 15. Interlobar effusion between all the lobes on the right side, the result of cardiac failure. Note the broad shadow of the interlobar fissure in the postero-anterior view.

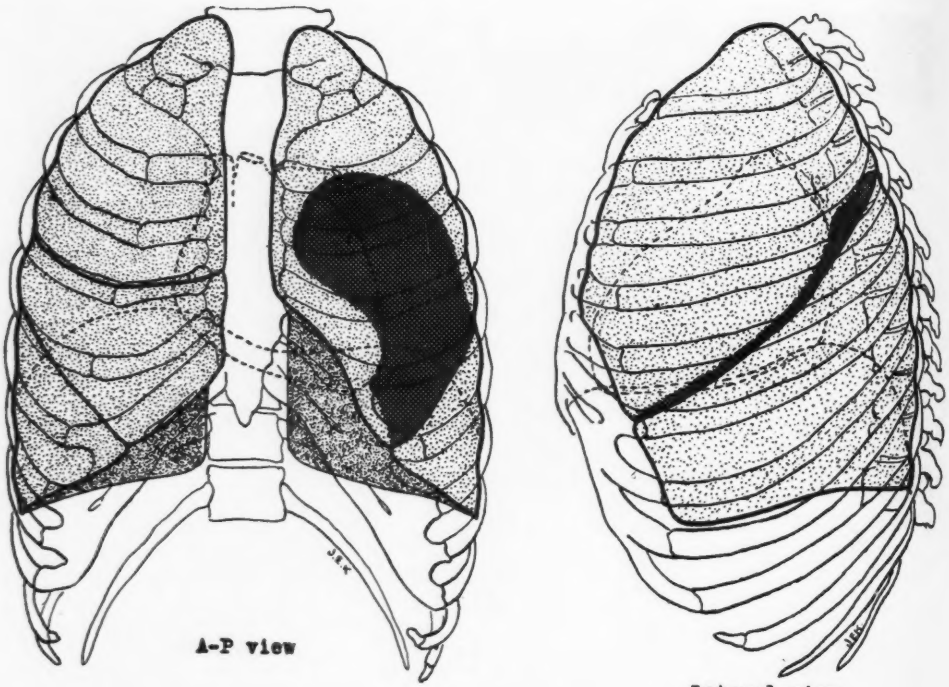


Fig. 20. Interlobar fissure between the left upper and left lower lobes.

Middle and Lower Lobes (Fig. 15).—Effusion between these lobes is the most frequent of all interlobar effusions. It is also the most interesting, as it is so often incorrectly diagnosed. A study of the postero-anterior view shows the similarity of the shadow of this fissure with the shadow of the middle lobe. (Compare Figures 2 and 15.) The oblique course of this fissure, clearly demonstrated on the lateral view, explains the reason for the broad shadow seen on the roentgenogram in the postero-anterior view.

COMMON ERRORS WHICH MAY BE MADE IN THE DIAGNOSIS OF EFFUSIONS BETWEEN THE MIDDLE AND LOWER LOBES

(1) *Atelectasis of the Middle Lobe* (Fig. 16).—Atelectatic lung is smaller than normal lung. The middle lobe is a small lobe and when atelectatic may diminish so in size as to approach the size of a large effusion between the lobes. Atelectasis of this lobe may be confused with an interlobar effusion.

(2) *High Diaphragm, Superimposed over Cardiac Shadow.*—This is a frequent error in diagnosis, and the overlapping oblique shadow, tapering at each end, has often been confused with an interlobar effusion between the middle and lower lobes. (See Fig. 17.)

Occasionally a pendulous breast shadow superimposed over the shadow of the diaphragm will give a similar oblique, tapered, dense shadow in the lateral view, which may be erroneously diagnosed interlobar effusion. The postero-anterior view will again rule out that diagnosis by the absence of the broad shadow in the lower chest extending outward from the hilus.

Right Interlobar Fissure Bordering the Upper Surface of the Lower Lobe (*The Major Fissure Separating the Lobes*) (Fig. 18).—This fissure separates the lower lobe from the upper lobe posteriorly (the hilus to the posterior chest wall), and the lower

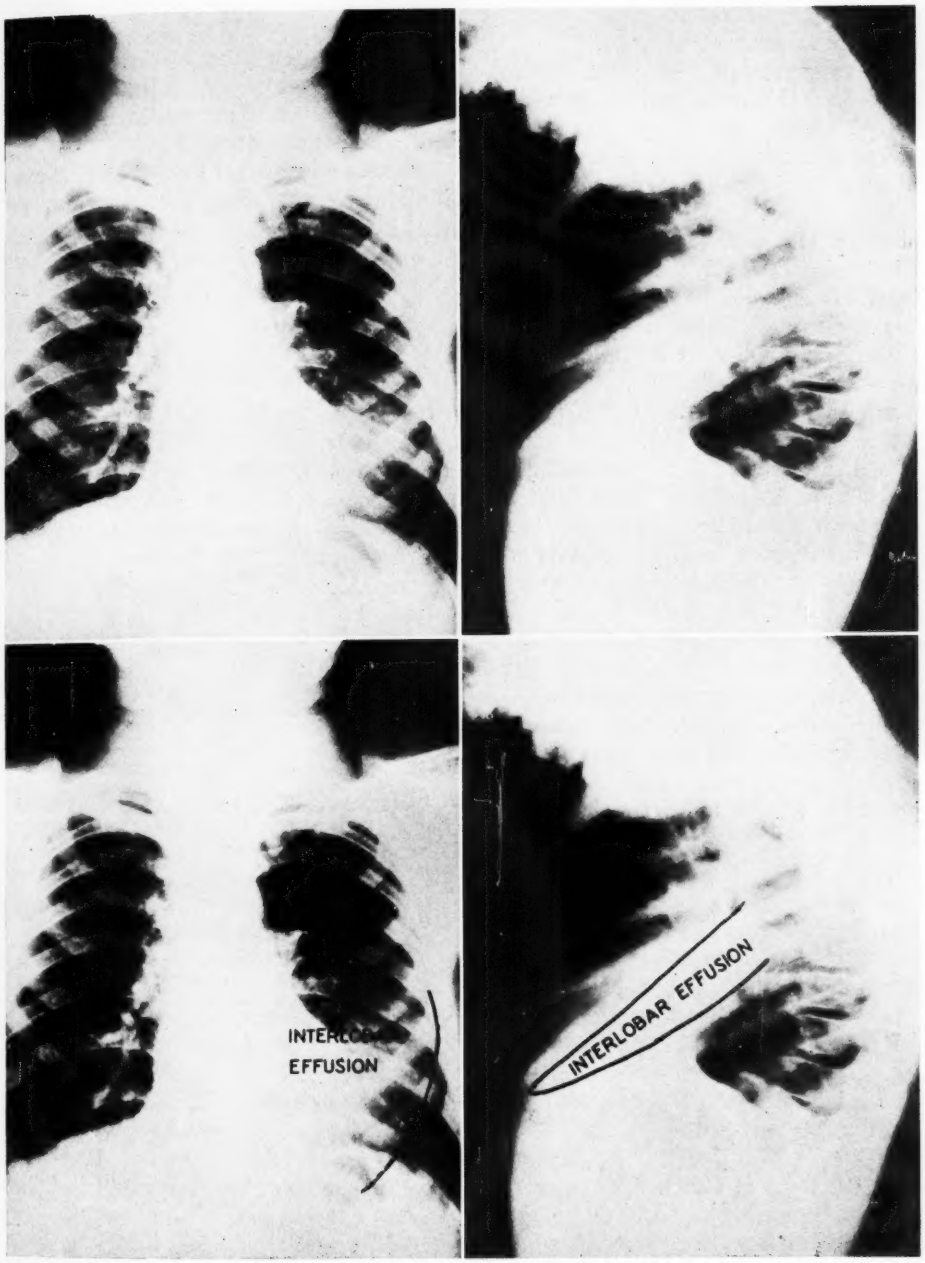


Fig. 20-*A*. Case 16. Interlobar effusion in anterior half of the interlobar fissure on the left side.

lobe from the middle lobe anteriorly (the hilus to the anterior chest wall).

The shadow of this fissure is a combination of the shadow (Figures 12 and 2) described above. In the postero-anterior view the shadow is broad, extensive, and bean-shaped. It can be easily mistaken for a large tumor. The lateral view clearly establishes the position of this shadow within the interlobar fissure.

Interlobar Effusion between All the Lobes on the Right Side (Fig. 19).—This shadow is a composite picture of all the interlobar shadows, as described above.

Interlobar Fissure between Left Upper and Left Lower Lobes (Fig. 20).—The shadow of this fissure is similar to the shadow on the right side, as described above (Fig. 18). It represents the anterior appearance of the surface of the lower lobe. Interlobar effusions involving this fissure are not common. If the posterior half of the fissure is involved, a shadow would be obtained comparable to the similar position on the right, *i.e.*, between the upper and lower lobes as described above (Fig. 12). Involvement of the anterior half of the fissure has a similar appearance to the fissure on the right side between the middle and lower lobes (Fig. 15).

SUMMARY

A set of diagrams is presented, illustrating the roentgenological appearance of the lobes of the lung and the interlobar fissures in the normal lung.

Roentgenograms of known clinical cases, illustrating the diagrams, are also presented.

Common errors in the diagnoses of some of these conditions have been pointed out.

CONCLUSION

We are indebted to Dr. A. L. Brown for suggestions and criticisms, and wish to thank Mr. J. Kelso, medical artist and photographer, for his assistance in the preparation of the diagrams and photographs.

516 Sutter St.

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A SURFACE LANDMARK CHART FOR USE IN X-RAY EXAMINATIONS OF THE TRUNK[1]

By WILLIAM EARL ANSPACH, M.D., *Chicago*

From the Division of Roentgenology, Department of Medicine, University of Chicago

THE ability to place patient, x-ray tube, and film in such relationship that some selected anatomical structure in the chest, abdomen, or pelvis shall appear at the center of the finished roentgenogram is an accomplishment much to be desired in a roentgenologist or his lay helper. Two general problems are involved: first, one must line up tube and film until the target lies perpendicularly above the center of the film; second, one must know what point on the skin of the patient's body lies directly above the anatomical structure selected for examination.

The first of these problems is more or less time-consuming if commercial types of apparatus are employed. In this particular laboratory it has been disposed of by equipping all exposing tables with special tube arms similar to the one on the obstetrical table described by Hodges and Ledoux.[1] When these tube arms are locked to the Potter grid and are made to stand at 90° to the table top, the target of the x-ray tube lies perpendicularly above the center of the film. The second problem is the subject of the present dissertation.

Sitting Height.—The sitting height of a subject is obviously closely related to the length of his spine and so this measurement was the first one used. Subjects were made to sit on a stool, their backs against a standard height board, and then, after sitting height had been measured, they were placed back down on the x-ray table. Measuring downward from the vertex along the anterior surface of the body in a plane parallel to the table top, a point was located halfway from crown to rump. The patient was now arranged on the table so that this $^1/_2$ S. H. (half sitting height) point lay along an imaginary perpendicular joining the target of the x-ray tube and the center of the film, and the exposure was made.

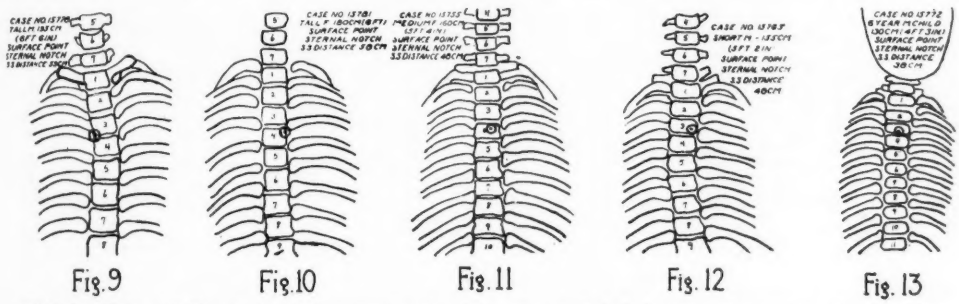

Fig. 9 Fig. 10 Fig. 11 Fig. 12 Fig. 13

Plate I. Tube and film centered at suprasternal notch. Dot and circle indicate film center. In these five subjects the notch corresponds closely to the body of the fourth thoracic vertebra.

In six adults ranging in build from short and fat to tall and lean, this one-half sitting height point was found to correspond to that portion of the spine bounded above by the center of the eighth thoracic vertebra, below by the center of the ninth thoracic vertebra. In one eight-year-old child it corresponded to the body of the eighth thoracic and in another, six years old, to the seventh thoracic vertebra. However, sitting height does not

[1] Abbreviated from a dissertation submitted to the graduate faculty of the University of Chicago in candidacy for the Degree of Master of Science.

Requests for reprints or other correspondence relative to this paper should be directed to the Division of Roentgenology, University of Chicago.

lend itself well to such work because the measurement cannot be made as the patient lies on the table ready for examination. spines, and iliac crests could not be palpated with sufficient precision. On the other hand, the suprasternal notch, which

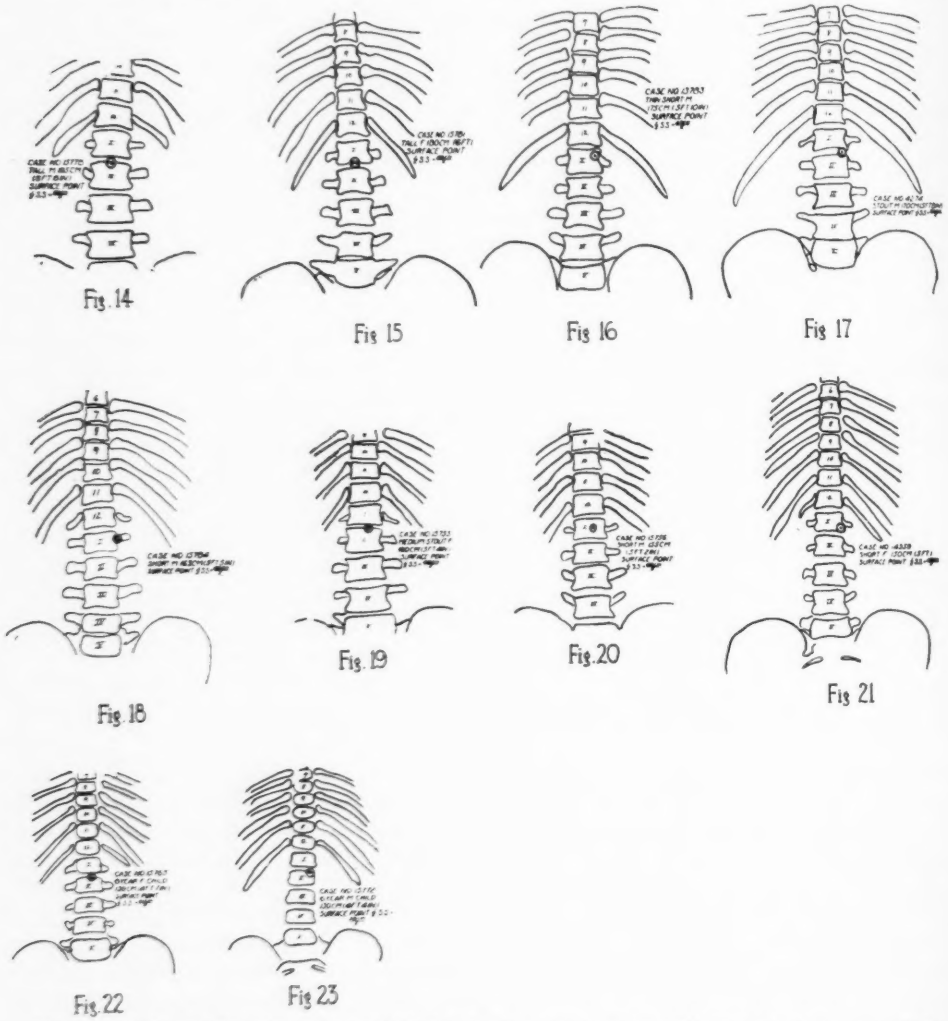

Plate II. Tube and film centered at middle of S. S. axis. In these ten subjects this point corresponds to the body of the first lumbar vertebra.

S. S. Axis (suprasternal notch—symphysis pubis axis).—Obviously the measurement of choice should be related to landmarks on the anterior surface of the body. Several such landmarks on examination proved unsatisfactory: the umbilicus did not have a constant relationship to spinal segments and the costal margin, iliac is always easy to locate regardless of age, sex, or build, was found to correspond closely to the fourth thoracic vertebra (Plate I), suggesting that this landmark might be used as the upper end of an axis.

A lower mark was now sought and after some study the superior margin of the symphysis pubis was selected as the best

one available. General experience has suggested that the symphysis might not be a dependable guide because presumably its repubis along the S. S. axis ($^3/_4$ S. S.) corresponded to the body of the fifth lumbar vertebra (Plate III), and $^1/_4$ S. S. lay directly

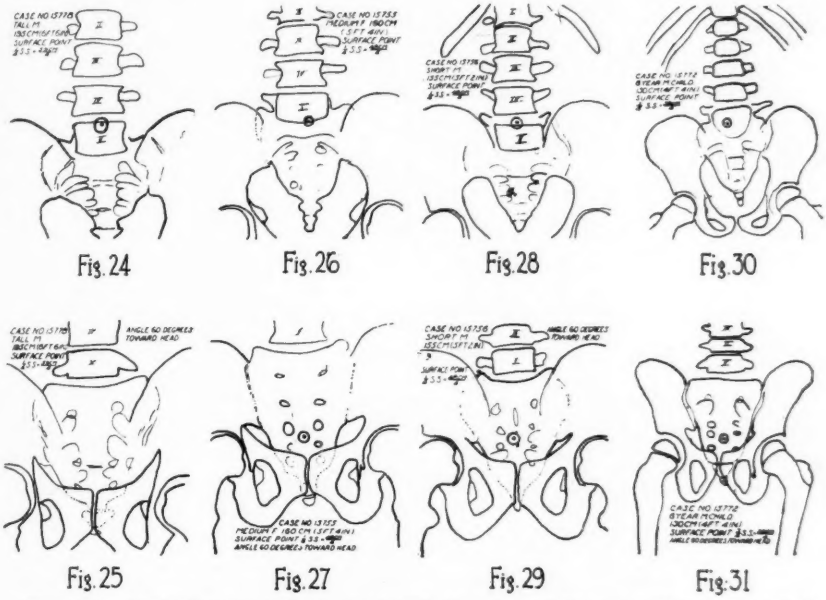

Plate III. In the four upper figures (24 to 30), the tube and film are centered on the S. S. axis one-quarter of the distance up from the upper surface of the symphysis pubis. With the radiation directed at 90° to the plane of the film, the body of the fifth lumbar vertebra appears at the center of the roentgenogram.

In the four lower figures (25 to 31), the film is centered as before at the one-quarter S. S. point but the tube is moved toward the subject's feet so that an imaginary line joining the center of the film and the center of the target makes an angle of 60° with the plane of the film. This angulation displaces skeletal structures upward, gives a better view of the sacrum, and brings the sacro-iliac joints to the center of the film.

lationship to neighboring structures would vary with sex and with the degree of inclination of the fifth lumbar on the first sacral vertebra. It was obvious also that it would fall below the lower end of the spine but this, of course, did not interfere with its use as the lower end of an axis. The "suprasternal notch—symphysis pubis axis" (abbreviated S. S. axis) is an imaginary line parallel to the surface on which the patient lies, tangent to the most prominent part of his anterior abdominal wall and terminated above by the suprasternal notch, below by the upper surface of the symphysis pubis. The middle of this axis ($^1/_2$ S. S.) was found to correspond to the body of the first lumbar vertebra (Plate II). A point one-quarter of the distance up from the symphysis above the ninth thoracic vertebra (Plate IV).

A Surface Landmark Chart.—It was now possible to construct a surface landmark chart (Plate V) showing the relationship between points on the S. S. axis and segments of the spine over S. S. axes ranging from 36 to 60 centimeters. This involves extrapolation below the observed values for some of the diagonals, but subsequent experience with the chart indicates that the extrapolation is sound. An S. S. axis of 36 cm. indicates a child approximately ten years old. Children younger than this seldom present much difficulty because their bodies are so small in comparison to the size of the film used that exact centering is of no great importance.

The chart is used as follows: With the subject on his back on the exposing table, the examiner palpates the superior margin of the symphysis pubis and of the sternal notch and marks these points on the skin. The straight-line distance between the two points is measured in a plane parallel to the table top and the measurement recorded. Suppose now it is desired to locate the point on the skin lying perpendicularly above the first lumbar vertebra. In the column at the left of the chart, one locates the end of the diagonal for the first lumbar vertebra and follows it downward and to the right to its intersection with the vertical line corresponding to the measured S. S. axis of the subject being examined. For instance, if the S. S. axis is 50, the diagonal for the first

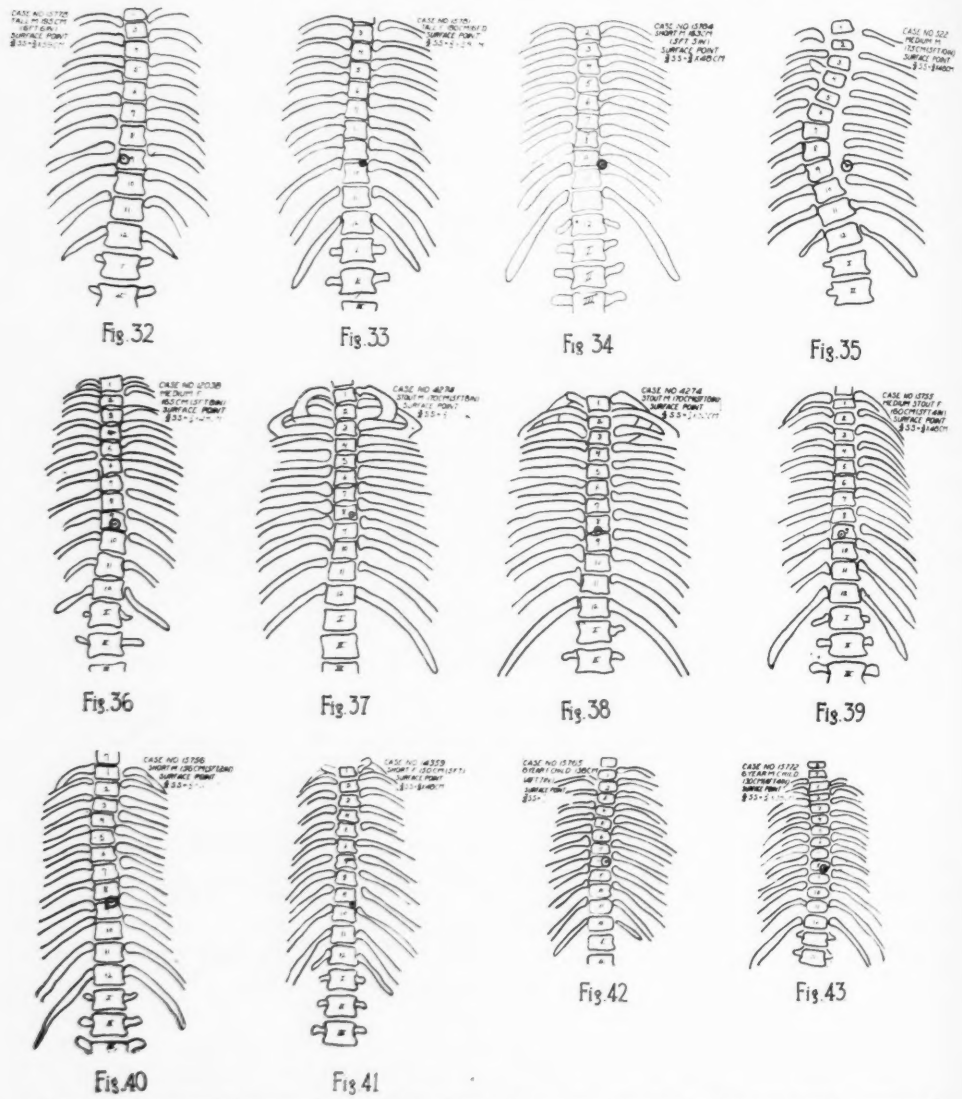

Plate IV. Tube and film centered at ¾ S. S. point. Film center (marked by dot and circle) corresponds closely to the body of the ninth thoracic vertebra.

TABLE I

Figure No.	Center Point	Portion of Spine Showing in Center of Film	
		As Predicted by Chart	As Actually Observed in the Roentgenogram
44	Suprasternal notch	Body of T 4	Lower surface of T 3
45	40 cm. above symphysis pubis	Lower surface of T 8	Upper surface of T 7
46	23 cm. above symphysis pubis	Body of L 2	Body of L 2
47	12 cm. above symphysis pubis	Body of L 5	Body of L 5
48	4.5 cm. above symphysis pubis	Pelvis	Pelvis
49	Same centering as in Fig. 48, but with smaller film to show only lower urinary tract.		

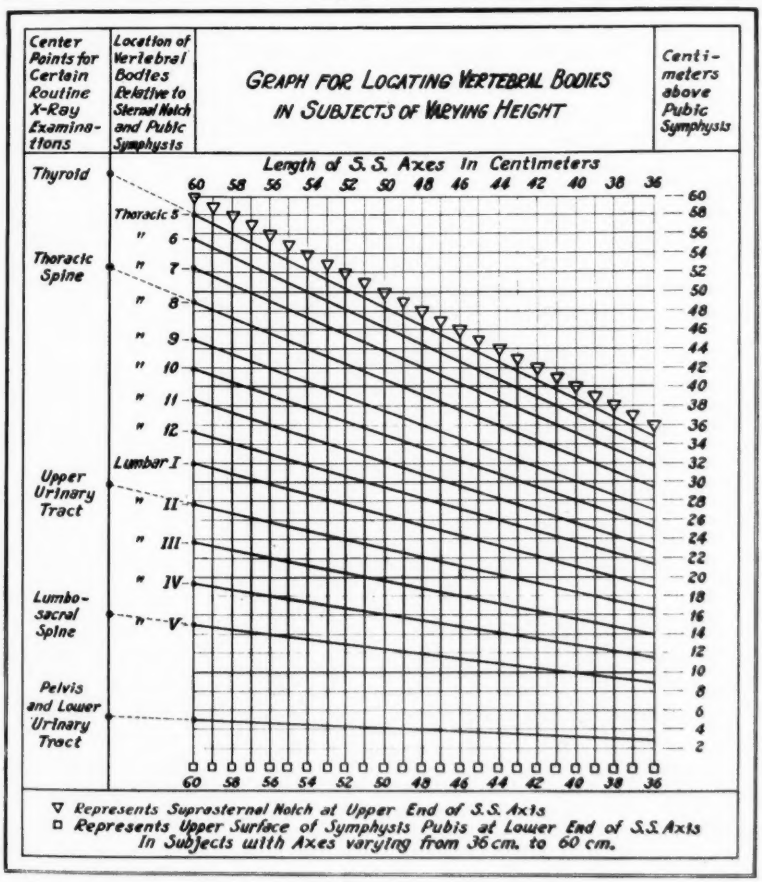
Center Points for Certain Routine X-Ray Examinations
Location of Vertebral Bodies Relative to Sternal Notch and Pubic Symphysis
GRAPH FOR LOCATING VERTEBRAL BODIES IN SUBJECTS OF VARYING HEIGHT
Centimeters above Pubic Symphysis
Length of S. S. Axes in Centimeters
60 58 56 54 52 50 48 46 44 42 40 38 36
Thyroid
Thoracic Spine
Upper Urinary Tract
Lumbo-sacral Spine
Pelvis and Lower Urinary Tract
Thoracic 5
" 6
" 7
" 8
" 9
" 10
" 11
" 12
Lumbar I
" II
" III
" IV
" V
60 58 56 54 52 50 48 46 44 42 40 38 36
▽ Represents Suprasternal Notch at Upper End of S.S. Axis
□ Represents Upper Surface of Symphysis Pubis at Lower End of S.S. Axis
In Subjects with Axes varying from 36 cm. to 60 cm.

Plate V.

lumbar vertebra will intercept it approximately 27 cm. above the symphysis pubis. Accordingly, if one measures upward from the symphysis pubis 27 cm. along the S. S. axis and makes a mark on the skin at this point, the mark will lie perpendicularly above the body of the first lumbar vertebra. If the S. S. axis is 40 and the spinal segment in question is the fifth lumbar, the mark will be made 15 cm. above the sym-

physis pubis; or if one wishes to locate the eighth thoracic vertebra and the subject has an S. S. axis of 48, the mark will be thoracic vertebra to the center of the film (theoretical value from chart, body of the eighth thoracic vertebra). Centering 23 cm.

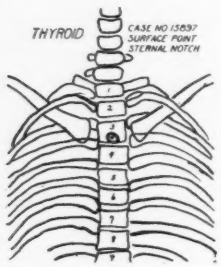

Fig. 44

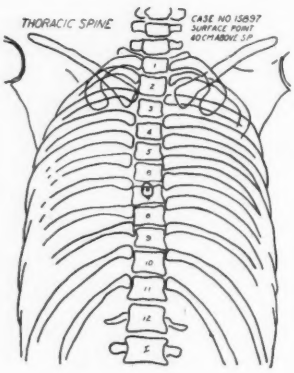

Fig. 45

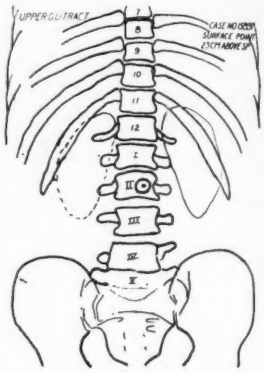

Fig. 46

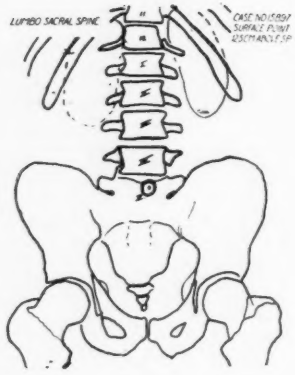

Fig. 47

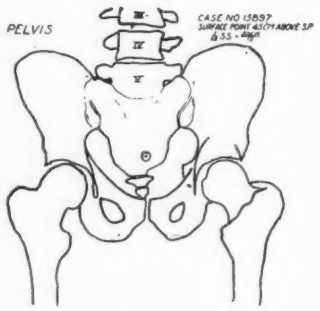

Fig. 48

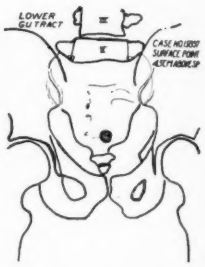

Fig. 49

Plate VI. Results obtained by use of chart in a subject 165 cm. tall and having an S. S. axis of 50 cm.

made 39 cm. above the symphysis pubis. For our own convenience there has been added a left-most column (Table I) showing the levels at which we commonly center for examinations of the thyroid, the entire thoracic spine, the upper urinary tract, the lumbosacral spine, the pelvis, and the lower urinary tract.

Plate VI illustrates the results obtained in an adult 165 cm. tall, whose S. S. axis measured 50 centimeters. Centering at the sternal notch (Fig. 44, Plate VI) brings the disc between the third and fourth thoracic vertebræ to the center of the film. Centering 40 cm. above the symphysis pubis (Fig. 45, Plate VI) brings the body of the seventh thoracic vertebra to the center of the film (theoretical value from chart, body of the eighth thoracic vertebra). Centering 23 cm. above the symphysis pubis (Fig. 46, Plate VI) places the second lumbar vertebra at the center of the film. The fifth lumbar vertebra lies directly beneath a point 12 cm. above the symphysis (Fig. 47, Plate VI); the pelvis is well placed if the film is centered 4.5 cm. above the symphysis (Fig. 48, Plate VI), and the same center point is satisfactory for 10 × 12 films of the lower urinary tract.

Oblique Views of Sacro-iliac Joints.—For special examinations of the sacro-iliac joints many workers place the subject on his back and angle the radiation 60° toward the head to compensate for the inclination of the sacrum and thus spread out

its image on the film. Centering for the examination is somewhat difficult because parts lying farther forward are projected strongly upward, due to the angulation of radiation. We find that, centering film and grid at the fifth lumbar vertebra and then angling the radiation 60° toward the head, the sacro-iliac joints usually appear at the middle of the completed film, regardless of the age or build of the subject.

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DUODENAL CARCINOMA: ITS RELATIONSHIP TO DUODENAL ULCER

By I. S. STARTZ, M.D., *Boro of Queens, N. Y.*

THE subject of carcinoma is always a vital consideration, especially in view of the fact that so little is known as regards its etiology and specific treatment, and that it has taken second place (3) as cause of death in our U. S. mortality statistics. And to go further, there is the realization that 75 per cent of all cancer cases occur in some part of the digestive system (6).

It is a surprising fact that two-thirds of the entire alimentary tract, namely, the small intestine, is almost immune from carcinomatous implantation. Primary carcinoma of the duodenum is a rarity. Eger (8), in collecting data from general autopsies totalling 350,286 cases, reports primary carcinoma of the duodenum in only 0.033 per cent of cases. Herman and Von Glahm (11), in their average incidence from autopsy combined figures by Max Müller, Perry and Shaw, Northnagel, Ruepp, Tieman, and Fritz Müller, quote a surprising and almost similar percentage of 0.035. It is customary to divide the duodenum into the first or supra-ampullary portion, the second or ampullary portion, and the third or infra-ampullary portion. Of the three divisions of the duodenum, 70 per cent of the cases reported (4) occur in the ampullary portion, 18 per cent in the supra-ampullary portion, and 12 per cent in the infra-ampullary portion. The duodenum is attacked almost as frequently as the jejunum and ileum combined.

The graphic chart (Fig. 1) has been compiled from average available statistics. It portrays more vividly the anatomic incidence of duodenal cancer in relation to other cancers of the gastro-intestinal tract, and to cancers as a whole.

Explanation Offered for Infrequency of Carcinoma of the Small Intestine.—Just why the small intestine should be relatively cancer-resistant, and the other parts of the alimentary tract, particularly the stomach, cancer-sensitive, is difficult to explain. The two contiguous portions of the pars pylorica and the duodenal cap do not present any sharp demarcation in the histologic appearance of the mucous membrane. Piersol's anatomy describes a "gradual" transformation in the structure of the mucous membrane between the two. Even the pyloric sphincter cannot always be recognized by the surgeon as the division line without utilizing the pyloric veins as a landmark (17). Nevertheless, the pyloric sphincter is the gate between the pre-pyloric cancer zone and the post-pyloric non-cancer zone.

Let us, therefore, discard the histologic study, and consider the matter from the physiologic viewpoint. The motility of the chyme is greater in the small intestine than in any other part of the gastro-intestinal tract. The average time required for the passage of food through the twenty feet of small intestine is four and three-eighths hours (13). The average meal in the stomach, while undergoing digestion, sojourns as long as from three to six hours in its migration from the cardiac end to the pyloric sphincter, a distance of eleven inches. The colon content gradually decreases in its motility from the cecum to the rectum. Correlating these facts with the incidence of gastro-intestinal cancers, there appears to be a direct relationship between the motility of the food and the frequency of carcinoma in the various portions of the gut. In other words, the predilection of primary carcinoma is in inverse proportion to the physiologic rate at which the food is propelled through the gastro-intestinal tract. On this basis, we can explain the large number of cases of carcinoma in the stomach, the rarity of carcinoma in the small intestine, and the reason for the incidence of

carcinoma increasing from the cecum toward the rectum (9).

It is logical to infer that the slower the material migrates through the gut, the greater opportunity there is for the production of chronic irritation or "wearing out" of the mucosa. This supports the generally accepted opinion that chronic irritation plays an important part in the causation of cancer.

Why Duodenal Ulcers do Not Become Malignant.—Based on the chronic irritation theory, it was believed that an ulcer was a favorable soil for carcinomatous implantation. However, this idea does not harmonize with actual facts. Although ulcers are very common in the duodenum, occurring three to four times as frequently as gastric ulcers (4), duodenal carcinomas are nevertheless extremely rare. Lichty (16) reports that "in a series of 486 cases of duodenal lesions, six lesions were found to be cancerous and 480 benign ulcers. In 780 cases of gastric lesions, 240 were found to be cancers and 540 benign ulcers. Apparently then the ulcer-bearing areas do not coincide in the duodenum as they do in the stomach." Judd (14) states that he was unable to find in the records of the Mayo Clinic any evidences that a primary carcinoma of the duodenum arose from an ulcer. Dewis and Morse (7) claim that duodenal carcinoma probably arises independently, and that little evidence has been found that it ever has arisen in a pre-existing ulcer. Schofield (21) informs us that duodenal ulcer has never been proven to be a predisposing cause of duodenal carcinoma. Hinton (12) asserts that, clinically, one can disregard the possibility of duodenal ulcer ever taking on malignant degeneration.

The case which I shall describe in the latter part of this paper reveals a primary carcinoma in the ampullary portion of the duodenum, independent of an associated ulcer in the supra-ampullary portion, which also favors the opinion that there is no connection between the two types of lesion. In my opinion, duodenal ulcers are not apt to become malignant for the reason that the majority of them are really "acute" and do not exist long enough to produce chronic irritation. These ulcers, which are small and superficial, are confined to the mucous membrane and will heal rapidly under medical treatment or no treatment at all (5). The recurrence of symptoms, following a period of freedom from distress, is generally due to the formation of a "new" ulcer at a different site. In the chronic ulcer, as originally described by Moynihan, there is destruction of the deeper racemose tubules and muscular coat, and this type of lesion may produce chronic irritation and become a favorable soil for carcinomatous implantation. The two cases presented by Arisz (1), in which callous duodenal ulcers have undergone malignant degeneration, belong to this category.

Symptoms and Physical Findings in Cases of Primary Carcinoma of Duodenum.—Although there are relatively only a few reported cases dealing with duodenal cancer, the writer has been impressed with the manifold detailed description of symptoms and physical findings that accompanied nearly all these articles. An effort is even made to portray a set of symptoms for each of the three sections of the duodenum. One is almost imbued with the idea that the diagnosis can be made from the symptomatology alone. Pain, nausea, vomiting, distention, malaise, loss of weight, diarrhea, melena, palpable abdominal mass, succussion splash, low red cell and hemoglobin, etc., may occur in any case of malignancy in the gastro-intestinal tract. The symptom of jaundice is emphasized by most of the writers as being present only in carcinoma of the ampullary portion of the duodenum (20). Absence of free HCl in gastric contents, and presence of occult blood following a meat-free diet are also considered important diagnostic features (19).

Roentgenologic Consideration.—Carman (4), in his second edition of "Roentgen Diagnosis of Diseases of the Alimentary Tract," published in 1920, states that cancer in the supra-ampullary area is not to be differentiated roentgenologically from duodenal ulcer. In describing the ampullary and infra-ampullary lesions, he asserts that

"the most important and constant x-ray finding is the gas and fluid distention of the bowel. The fluid may show hydroscopic

Mills (18), in his classic paper on "Small Intestinal States," published in 1922, brought into focus numerous lesions of the

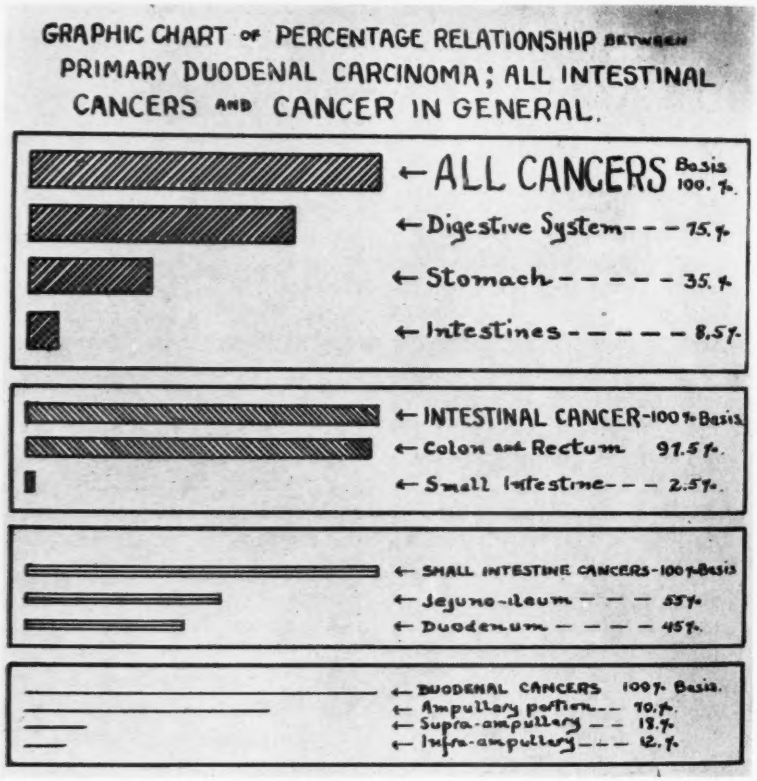
GRAPHIC CHART OF PERCENTAGE RELATIONSHIP BETWEEN PRIMARY DUODENAL CARCINOMA; ALL INTESTINAL CANCERS AND CANCER IN GENERAL.
← ALL CANCERS Basis 100. %.
← Digestive System - - - 75. %
← Stomach - - - - - 35. %
← Intestines - - - - - 8.5%
← INTESTINAL CANCER-100% Basis
← Colon and Rectum 97.5%.
← Small Intestine - - - 2.5%.
← SMALL INTESTINE CANCERS-100% Basis
← Jejuno-ileum - - - 55%
← Duodenum - - - - 45%.
← DUODENAL CANCERS 100% Basis.
← Ampullary portion - - - 70.%.
← Supra-ampullary - - 13.%.
← Infra-ampullary - - - 12.%.

Fig. 1.

levels in the various loops [Figs. 2 and 3]. The amount of gas, fluid, and distention will depend on the location of the lesion; it is greatest in the low obstruction." Herman and Von Glahm (11), in 1921, restudied the roentgenograms of a case of primary carcinoma of the supra-ampullary portion after the autopsy diagnosis had been made, and noticed a slender canal or "stalk" representing the stream of barium passing through the lumen of the duodenal growth. They believe that "a similar roentgen-ray picture in the future will be an aid to roentgenologists in the diagnosis of carcinoma of the supra-ampullary portion of the duodenum."

small intestine that can be diagnosed roentgenologically. The following extract is worthy of note: "Any organic process involving the small intestinal wall, either primarily or secondarily, will modify the x-ray shadow of the contents of the part involved and thus render direct diagnostic evidence of its presence, if not as strikingly as in the instance of stomach and colon or yet as suggestively as to the nature of the lesion, none the less positively as to an organic pathologic process being present." Accompanying this manuscript are numerous illustrating roentgenograms, but the author does not venture to make a definite diagnosis other than that of an "organic

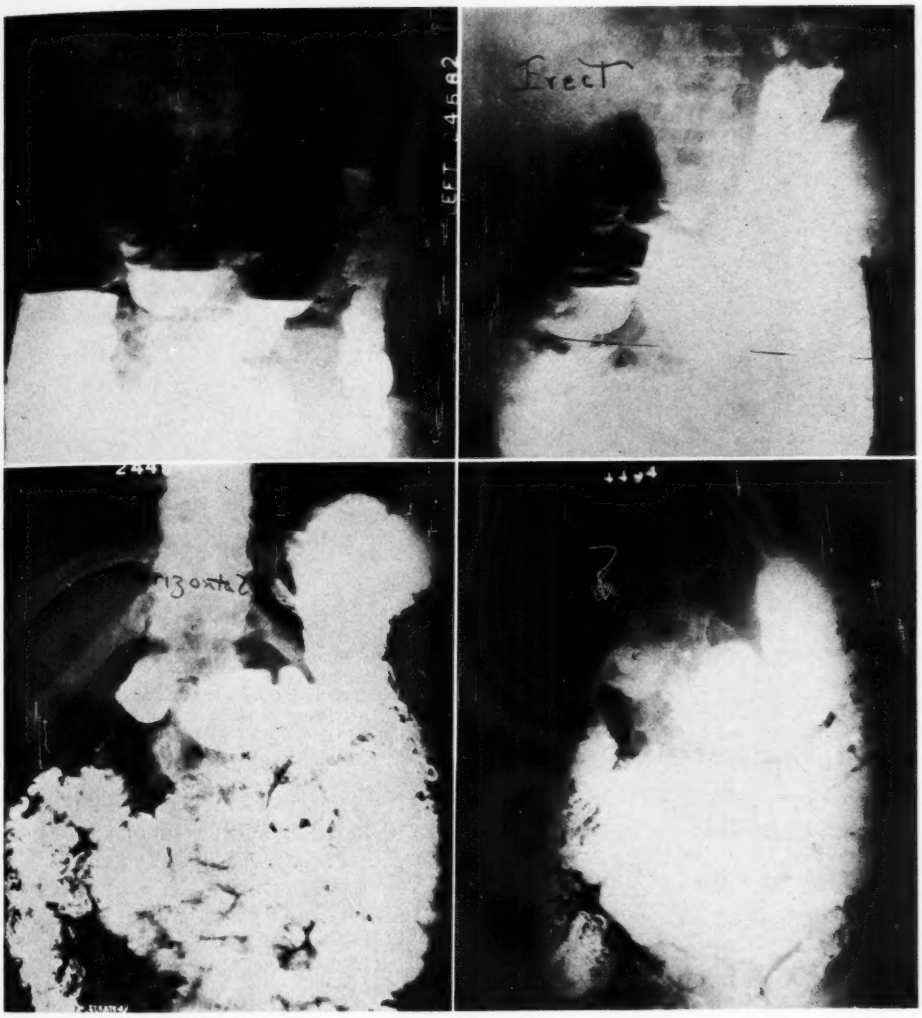

Fig. 2 (*upper left*). Roentgenogram made in upright posture, showing hydroscopic levels in the distended intestinal loops due to stasis, caused by a carcinoma of the upper descending colon near the splenic flexure.

Fig. 3 (*upper right*). Roentgenogram in upright position, showing multiple fluid level pockets within distended loops of the small intestine in the right upper quadrant, due to adhesions. Hydroscopic fluid levels may occur in any case of intestinal obstruction, due to a lesion in the small or large intestine.

Fig. 4 (*lower left*). Full stomach film, showing perfectly filled stomach and duodenal cap. The small intestinal pattern (except for the ampullary portion) presents a normal appearance.

Fig. 5 (*lower right*). Barium-filled stomach in erect position. No hydroscopic fluid levels seen within small intestinal coils. The duodenal ulcer is faintly visualized.

lesion in the small intestine." William H. Stewart, in discussing this paper at its presentation before the American Roentgen Ray Society, stressed the importance of having patients x-rayed at frequent intervals between the time the stomach is filled and the six-hour study.

Eusterman, Berman and Swan (10), in their paper published in 1925, report a careful analysis of 15 cases of duodenal carcinoma. Roentgen-ray examination was accomplished in 10 of these cases. In three cases, the roentgen findings were negative. In five cases, there was present a dilated

stomach with considerable barium residue without a demonstrable lesion. In two cases, the "demonstration of a large stomach, dilated duodenum, and retention made an exact anatomic diagnosis possible."

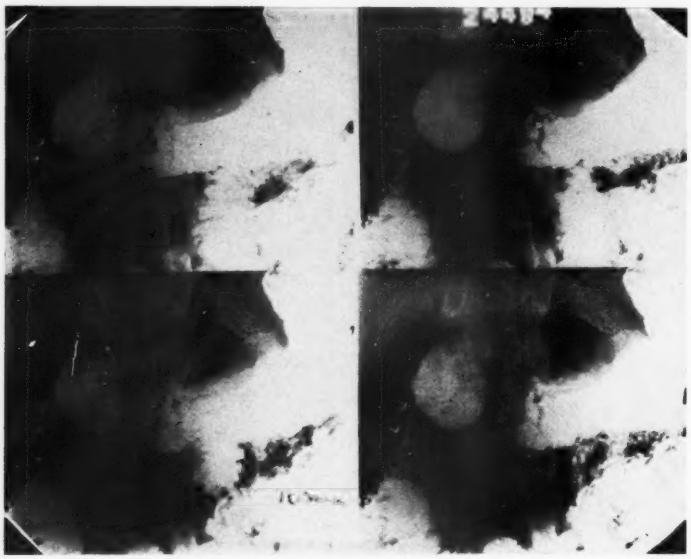

Fig. 6. Serialogram taken 10 minutes after the barium meal was swallowed. The string-like narrowed lumen of the ampullary portion of the duodenum is constant in all of the exposures. There is too much barium within the duodenal cap to visualize the ulcer.

Soper (22), in 1929, emphasized the importance of making the exposures in the erect position. He also impresses us with the fact that primary carcinoma of the small intestine may simulate a duodenal diverticulum. In the former, there is generally a gas bubble surmounting the fluid level: its outline is of irregular character, the shadow is apt to be evanescent, and the occult blood reaction is positive. Among the lesions portrayed in the roentgenographic reproductions accompanying the article are three cases of malignant tumors of the small intestine. A pre-operative diagnosis of malignancy of the small intestine was made in two of these cases, based on x-ray findings and strong persistence of occult blood.

A Utopian publication is that of the Cole Collaborators (5) in their book titled "Radiographic Exploration of the Mucosa of the Gastro-intestinal Tract," published in 1934. They state that "pathologic lesions of the small intestine have been considered to be so infrequent as compared with those of the stomach, cap, and colon, that they have not attracted the roentgenologist's attention. We have learned from bitter experience that unless one looks for these lesions he does not see them, even though there are definite roentgenologic findings present." In another chapter, "Four Fundamental Findings" for diagnosis of carcinoma are applied to the small intestine, namely, first, constant filling defect, second, loss of mucosal folds, third, loss of pliability of wall, and fourth, abnormal appearance of mucosal pattern. Cole mentions only two cases of neoplasm of the small intestine. One case was pre-operatively diagnosed as dilation of the duodenum caused by a small annular constriction about one or two centimeters in length. Biopsy disclosed a sarcoma at the duodenal-jejunal junction. The other case shown "was evidenced roentgenographically by a

small annular area of constriction about two centimeters in length. The lumen of the gut was constant in size and shape, and the irregularities of the surface are similar to those observed in carcinoma of the colon. The lesion was part of a general peritoneal carcinomatosis, and whether this was the primary lesion could not be determined at operation. The local lesion was not removed and, therefore, the gross or microscopic sections are not available." In Cole's book there is also incorporated a roentgenographic print of a carcinoma of the small intestine, interpreted as such by C. T. Baker.

I wish to report the following unusual and rare case of duodenal ulcer of the supra-ampullary portion of the duodenum, combined with an independent primary carcinoma of the ampullary portion of the duodenum, both of these lesions being clearly visualized in the roentgenograms (Figs. 4, 5, 6, 7, 8, and 9).

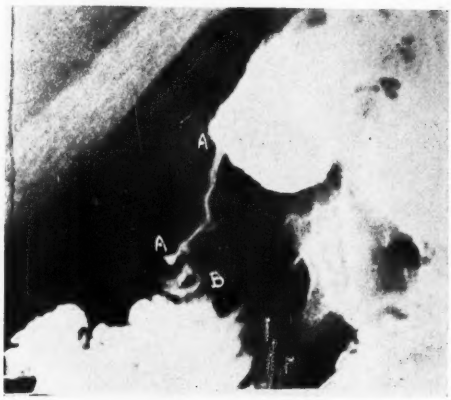

Fig. 7. Roentgenogram showing the ampullary portion of the duodenum magnified. The region *A–A* presents a constant string-like contour defect, with absence of valvulæ conniventes. The valvulæ markings of the infra-ampullary portion marked (*B*) are clearly visualized.

CASE REPORT

N. C., a white male, aged 34 years, a chauffeur by occupation, was referred to my office by Dr. H. Rappaport for roentgenologic study of the alimentary tract. His family history, past history, and habits were unessential.

Present Complaint.—The patient had experienced an insidious onset about one month before, with pain in the epigastric region one or two hours after eating, which was relieved by food and bicarbonate of soda. For the past two weeks he had suffered with "heart burn," with an acid taste in his mouth. He had lost 22 pounds in the past two months. He was constipated; he had no black stools. He had had vomiting attacks during the past ten days.

COPY OF X-RAY REPORT, AUG. 5, 1935

Esophagus.—The barium paste passes readily through the esophagus, meeting with no delay or obstruction. There are no evidences of cardiospasm, peptic esophagitis, or any organic filling defect.

Stomach.—Not enlarged, orthotonic, in normal relation to the spine, and peristaltic activity is increased. Greater and lesser curvatures do not show any constant deformity or filling defect. Greater curvature is 4.5 inches above intercrestal line in horizontal posture, and drops to 1.75 inches above intercrestal line in the erect position.

Duodenum.—The duodenal cap shows perfect filling ten minutes after swallowing the barium meal, but after a period of one-half hour, with "dosified expression and compression," a large ulcer is seen *en face*, apparently within the posterior wall. The ampullary portion of the duodenum presents a narrow string-like appearance, the same having a constant contour defect in all of the serial exposures. No valvulæ conniventes are seen in this portion of the duodenum. No palpable mass or fixation is present in the first or second portions of the duodenum, but there is a moderate amount of tenderness. No hydroscopic fluid levels or dilated small intestinal coils are seen in the erect position.

Three-hour Study (Fluoroscopy Only).—No additional information was obtained.

Six-hour Study.—A fleck of barium is retained in the crater of the duodenal ulcer. The stomach is empty. The barium meal is distributed throughout the large intestine and pelvic colon.

X-ray Diagnosis.—(1) Ulcer of first por-

tion of the duodenum (not associated with pyloric obstruction). (2) Severe duodenitis of second portion of the duodenum, first portion of the duodenum. All of the four fundamental findings as depicted by Cole (5) are present in the ampullary portion.

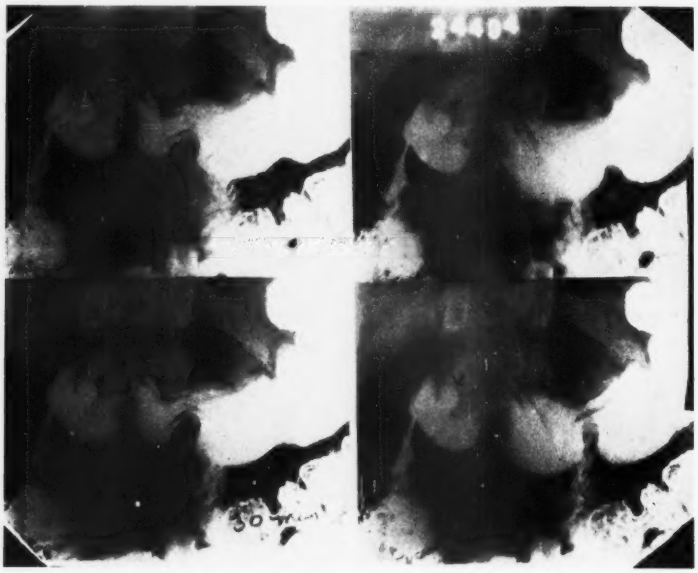

Fig. 8. Serialogram taken 30 minutes after swallowing the barium meal, shows *en face* duodenal ulcer. The narrowed ampullary portion of the duodenum is persistent.

with possibility of a carcinoma of this portion of the duodenum.

COMMENT ON X-RAY REPORT

Based on the x-ray findings, and with a history of considerable loss in weight, the writer at the time of the interpretation of the roentgenograms was convinced that he was dealing with a duodenal carcinoma in addition to a duodenal ulcer. However, due to the rarity of this combination, and in order to avoid the criticism of having made a fanciful roentgen-ray diagnosis, he lacked the courage of his conviction and reported the x-ray diagnosis as stated above. The opinion of "severe duodenitis" of the second portion was based on a similar string-like appearance seen in cases of ileitis (13) involving the terminal ileum. This also closely resembles the "stalk-like" barium shadow described by Herman and Von Glahm (11), in a case of carcinoma of the

Subsequent Progress of the Patient.—On Aug. 5, 1935, the day following completion of the x-ray examination, the patient went into shock, was rushed to the New York Hospital, and admitted to the service of Dr. DuBois. Two blood transfusions were given with apparently very little beneficial effect. The patient died at 7 P.M. the following day.

EXTRACTS FROM AUTOPSY REPORT

Autopsy was performed by Dr. Hall, on Aug. 6, at 8:20 P.M. (Autopsy No. 8219.)

Anatomical Diagnosis.—Chronic duodenal ulcer; rupture of branch of superior pancreatico-duodenal artery, with hemorrhage into stomach and intestines, and carcinoma of the greater duodenal papilla, with metastasis to the regional lymph nodes and liver.

GROSS EXAMINATION

Gastro-intestinal Tract.—Esophagus is normal. The stomach contains a large amount of dark brownish-red fluid. The lining mucosa is normal. Two centimeters below the pyloric ring on the posterior mucosal surface of the duodenum, there is a large crater, 2.5 cm. in diameter, which extends down into the muscularis of the duodenum. The walls of the crater are firm in consistency and grayish-red in color; the area surrounding it is light red in color. The floor is grayish-red. Running across the floor of the crater are two blood vessels, the superior walls of which have been eroded through. The ampulla of Vater projects into the duodenal lumen and is slightly firmer in consistency than normal. On cut section through the ampulla of Vater, it is firm and grayish-white in color. The rest of the gastro-intestinal tract contains a large amount of brownish-red material.

Pancreas.—Is normal in shape and position: the head is much firmer and more resistant than normal. The lymph nodes at the head of the pancreas are from 0.5 to 1.5 cm. in diameter, and are very firm in consistency. Section through these nodes shows them to be firm, grayish-white in color. On cut section through the head of the pancreas, there is seen a small 1 × 3 cm. grayish-white area located against the posterior wall of the first portion of the duodenum. The rest of the pancreatic tissue shows the normal architecture. The region of the ampulla of Vater is slightly firmer in consistency than normal.

Liver.—Is slightly irregular in shape, and weighs 1,640 grams. Scattered over the entire surface of the liver are numerous firm, light brownish-white nodules, 1 to 15 mm. in size, surrounded by a narrow band of dark red. The rest of the liver surface is a normal light brownish-red color. On cut section, scattered throughout the entire liver tissue, are numerous light tannish-white areas, 1 to 17 mm. in size, surrounded by a thin margin of dark red. The central portions of these areas are very friable and somewhat red-tinged. The remaining liver tissue is light brown in color.

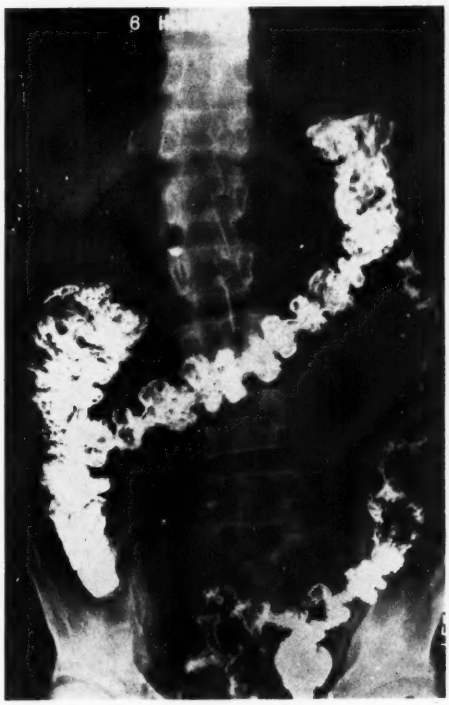

Fig. 9. Six-hour study, showing a fleck of barium retained within the crater of the duodenal cap.

MICROSCOPIC EXAMINATION

Esophagus.—Section transversely across the esophagus shows areas in which the stratified epithelium is missing, and the mucosa contains a moderate number of polymorphonuclear and mononuclear leukocytes.

Duodenal Ulcer.—Section through the wall and a portion at the floor of the ulcer shows the overhanging wall to consist of a dense fibrous connective tissue, in which are an accumulation of polymorphonuclear and mononuclear leukocytes in the submucosa and muscularis. The adjacent mucosa is normal. The floor of the ulcer consists of a slight amount of fibrous tissue, but most of the structure is necrotic. The floor of the ulcer shows some dense bands of fibrous tissue penetrating into the adjacent pancreatic tissue.

Duodenum at Ampulla of Vater.—The mucosa of the duodenum up to the ampulla is intact. The submucosa down to the muscularis consists of dense connective tissue, in which are groups and columns of moderate sized cells similar to those found in the liver tumor. The tumor tissue is separated entirely from the pancreas and has no relationship with the bile duct that is seen in this section.

Liver.—In the liver, there are several circumscribed areas consisting of cells of moderate size that have an acidophilic staining cytoplasm, which in most instances do not have a definite outline, containing round and oval, mostly dark staining peripherally placed nuclei. However, a few of the nuclei are vesicular. For the most part, the cells are packed in groups, with no definite arrangement except in several areas in which the cells radiate around delicate connective tissue cords in which there is usually a small blood vessel. There are a moderate number of mitotic figures. One large area of the tumor tissue consists of a homogeneous dark acidophilic material, in which are seen a moderate number of dark staining and pyknotic nuclei. The surrounding liver tissue is compressed, and the sinusoids are filled with red blood cells.

Lymph Node at Head of Pancreas.—This section consists of areas composed of dense bands of connective tissue, between which are groups of tumor cells similar to those described under "Liver" and areas in which there are mostly tumor cells with little connective tissue showing. In one large area, the connective tissue divides the tumor tissue into small cords and groups of cells. The adjacent portion of pancreatic tissue is similar to that already described, except that several of the islets show a slight amount of hyalinization.

Pancreas.—One section of the pancreas shows an increase in both inter- and intralobular connective tissue. In the interlobular connective tissue septa, there is a slight increase in lymphocytes and large mononuclear cells. The other section of the pancreas, taken against the base of the ulcer, consists of a thick layer of connective tissue in which are a few lymphocytes and large mononuclear cells. The rest of the section is composed of pancreatic tissue in which there is an increase in fibrous connective tissue.

DISCUSSION

This case is of unusual interest because of the following:

(1) Rarity of duodenal carcinoma.
(2) Its independent association with a duodenal ulcer.
(3) The fact that both lesions are clearly demonstrable in the roentgenograms.
(4) Patient died from internal hemorrhage—an erosion of a blood vessel within the duodenal ulcer.
(5) There were no signs present of intestinal obstruction.
(6) Patient was 34 years old. (Average age of cases previously reported is between 60 and 70 years.)
(7) Metastasis was present to adjacent lymph nodes and liver. (Brill (2) states that metastasis even to the contiguous lumph nodes is not common.)
(8) Patient was not jaundiced. (Eger states (8) that in cases of carcinoma of ampullary portion of duodenum, jaundice generally appears early.)

ESSENTIALS FOR THE PRE-OPERATIVE DIAGNOSIS OF SMALL INTESTINAL NEOPLASM

As a result of this general survey, we should be impressed with the following important facts as to one's ability to diagnose primary neoplasm of the small intestine.

(1) The roentgenologic study, *if properly performed*, is the strongest link in the chain of our diagnostic methods. The roentgenologist who follows the orthodox routine examination of the gastro-intestinal tract by studying the full stomach and then permitting the long interval of six hours to go by before doing the second examination, is sadly neglecting the investigation of two-thirds of the entire alimentary tract. To him, the small intestine is the "silent" or "blind" area. Abnormalities in the small intestinal pattern are best seen from the

two and one-half to six-hour period following the meal, and exposures should be made at frequent intervals. The erect position is valuable for the demonstration of fluid levels within the intestinal coils, which are frequently associated with partial intestinal obstruction.

(2) The well trained roentgenologist should be capable of detecting an "organic" lesion in the small intestine. However, an attempt to specify the exact nature and site of the lesion is often a shrewd guess and may result in a fantastic x-ray diagnosis out of gear with that of the autopsy report.

(3) The presence of symptoms and physical findings suggesting gastro-intestinal malignancy (including absence of free HCl in gastric contents, and presence of occult blood in stools), *plus* the recognition of a small intestinal organic lesion roentgenologically, should spell a pre-operative diagnosis of carcinoma of the small intestine.

(4) Remember that the *correct* diagnosis of carcinoma of the small intestine has heretofore in the majority of cases been made either during the exploratory biopsy or at the postmortem examination. However, it is hoped that this symposium will encourage the roentgenologist to *look for* small intestinal lesions so that the percentage of cases diagnosed pre-operatively will be materially increased.

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CONSIDERATIONS OF THE VARIABLE RECOVERY FACTOR OF TISSUE

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ABSTRACT

It is assumed that the recovery factor is some function of the fraction of the total number of cells which are undamaged by n quantum "hits": $Rf(S/N)$. The fraction S/N is assumed to be given by the probability distribution expression:

$$\frac{S}{N} = e^{-\lambda q}\left\{1 + \frac{\lambda q}{1!} + \frac{(\lambda q)^2}{2!} + \frac{(\lambda q)^3}{3!} + \frac{(\lambda q)^4}{4!} + \cdots \cdots + \frac{(\lambda q)^{n-1}}{(n-1)!}\right\}$$

which has been tested by extensive experiments on various biological objects. The fraction S/N is determined after each dose in the protracted dose technic, and the recovery factor calculated on the simple assumption that it is directly proportional to S/N. Cumulative dose curves are given.

WHEN tissue is subjected to a series of doses of radiation given at regular intervals, it is frequently desirable for the radiologist to know how much dose has accumulated after a given time. Thus Reinhard and Goltz (1) have given values of the cumulative dose for different increments of dosage utilizing a recovery factor of 8 per cent per day, as determined by Stenstrom and Mattick (2) for x-rays of 0.16 Å. effective wave length. Hoffman and Reinhard (3) have given methods of calculating cumulative dose in the protracted method of radiation over long periods of time. These workers, however, assumed that the recovery factor remained constant over the time of treatment.

Now, it is not unreasonable to suppose if a tissue is exposed to repeated assaults such as it receives in the protracted dosage technics, with the treatment extended over a long period of time, that its power of recovery will be diminished until eventually its constituent cells are powerless to overcome any radiation effects. When a cell is dead, the cumulative dose is equal to the total dose administered; in other words, we are putting radiant energy into an inert physico-chemical system.

As has been suggested by Holthusen (4), the process of recovery from radiation effects consists of two possible processes: in the first, the photo-chemical products formed are turned into their original compounds by a reversible chemical reaction; in the second, the photo-chemical products are removed from the tissue by mechanical means, presumably diffusion. One may think of this process of recovery from radiation effects as a "washing away" of the undesirable photo-chemical products. For the sake of generality, it should be added that by photo-chemical products we mean any type of molecule which has been put into an energy state other than that which it would have in the undisturbed tissue. This would include molecules, electrons of which are vibrating in higher energy states, or constituent atoms of which are in other than normal energy states, or those that have lost a photo-electron, etc.

We will now calculate the cumulative dose, D_c, for the general case. Suppose that the increment of dose is D and that it is administered at regular intervals in time. The fraction of the dose remaining in the tissue after the first interval will be k_1D, where $0 < k_1 < 1$. The second dose is administered, and after the second interval the remaining dose is

$$D_c = \{Dk_1 + D\}k_2 \qquad 0 < k_2 < 1 \tag{1}$$

After the third interval we get.

$$D_c = \{\{Dk_1 + D\}k_2 + Dk_3 = Dk_1k_2k_3 + k_2k_3 + k_3\} \tag{2}$$

and after the j^{th} dose:

$$D_e = D\{k_1k_2k_3 \ldots k_j + k_2k_3 \ldots k_j + k_3k_4 \ldots k_j + \ldots \ldots + k_j\} \quad (3)$$

This is a perfectly general expression for the cumulative dose. We have put no special conditions on the k's except that they be greater than zero and less than one. When k_j is equal to unity, it means that the tissue is unable to overcome radiation effects. It is conceivable that the first few k's might be equal to zero, if the dose D is very weak. The radiation effects are so small that the effects of the first few doses are completely washed away in the interval between doses. It must be emphasized, however, that each set of k's depends on the size of D and the length of the interval between doses. In other words, each scheme of administering dosage will elicit a different reaction from the tissue. The k's are a function of D and of the time interval T. We may write the functional relationship as:

$$k_1k_2k_3 \ldots k_j = f(D, T) \quad (4)$$

In order to discuss the variation of the k's, it will be necessary to make certain assumptions about the power of recovery of tissue. The power of recovery will be dependent on the fraction of the total number of cells that are alive. There may be a complicated relation between this fraction of live cells and the recovery factor. For simplicity of calculation we shall assume in this discussion that the power of recovery is directly proportional to the fraction of the live cells in a tissue. If R is the coefficient of recovery, and S is the number of live cells out of a total of N cells, we have

$$R = c\frac{S}{N} \quad (5)$$

where c is a proportionality constant. The fraction of dose remaining in the tissue will be

$$k = 1 - R = 1 - c\frac{S}{N}. \quad (6)$$

Thus when $S = 0$, $k = 1$, and all of the energy of the dose, D, remains in the tissue. When $S = N$, that is, when all cells are alive, $k = 1 - c$, and the power of recovery is at a maximum.

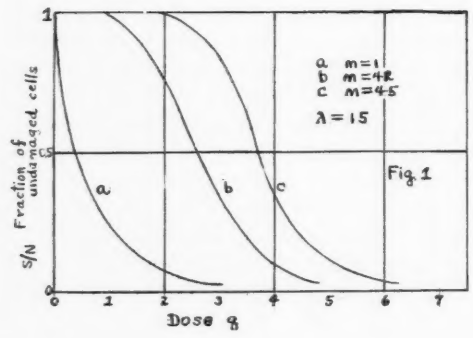
We need not, however, think in terms of the live cells. We may use the fraction of cells which have not yet been "hit" by n photons. In order to calculate the fraction S/N we may utilize the statistical methods developed by Blau and Altenburger (6) and by Crowther (5). Suppose an idealized slice of tissue is irradiated by a beam of photons incident perpendicularly on the plane of the tissue. Considering the cells as independent individuals, the number of cells not yet hit by n photons will be, according to Crowther,

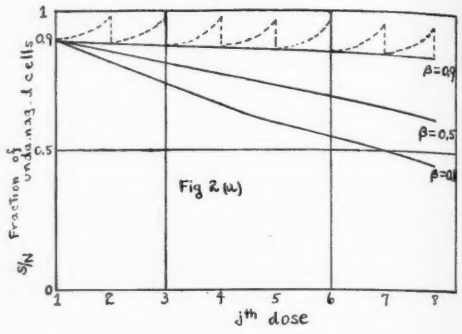
$$S = Ne^{-\lambda q}\left\{1 + \lambda q + \frac{\lambda^2 q^2}{2!} + \frac{\lambda^3 q^3}{3!} + \ldots \ldots + \frac{(\lambda q)^{n-1}}{(n-1)!}\right\} \quad (7)$$

where λ is the probability of a hit for unit dose, and q is the dose, which may be written in terms of beam intensity, I, and time, t, as $q = It$. Since tissue is, by definition, a more or less homogeneous aggregate of cells, we may think of an average number of hits, h, required to damage a cell. If such an average for a given tissue is possible, Equation (7) is modified according to Zuppinger (and Meissner) (8) as

$$S = Ne^{-(\lambda q + h)}\left\{1 + \lambda q(1 + h) + \frac{(\lambda q)^2}{2!}\left(1 + \frac{h}{1!} + \frac{2!}{h^2}\right) + \frac{(\lambda q)^3}{3!}\left(1 + \frac{h}{1!} + \frac{h^2}{2!} + \frac{h^3}{3!}\right) + \ldots\right\} \quad (8)$$

an expression which has been discussed by Braun and Holthusen (7). Equation (7) has received ample verification by the experiments of Packard (9) on *Drosophila* eggs, Braun and Holthusen's work on *Ascaris* eggs (7), Zuppinger's work on *Ascaris* eggs (8). Canti and Spear (10) give curves for the decrease of the number of mitotic figures in tissue culture *in vitro* after irradiation with gamma rays, which have the general shape of the curve S/N





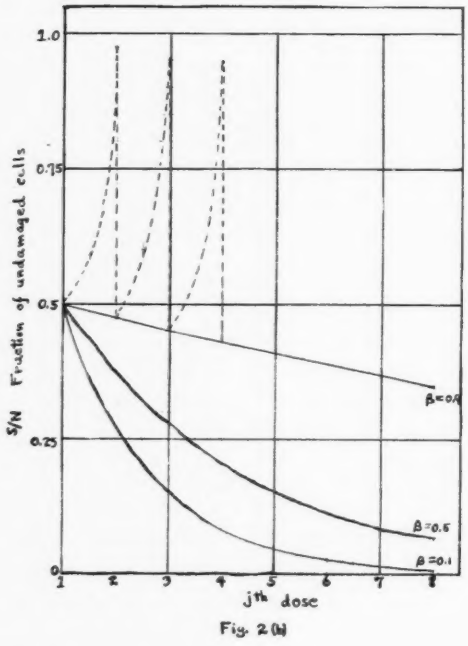


Fig. 2(b)

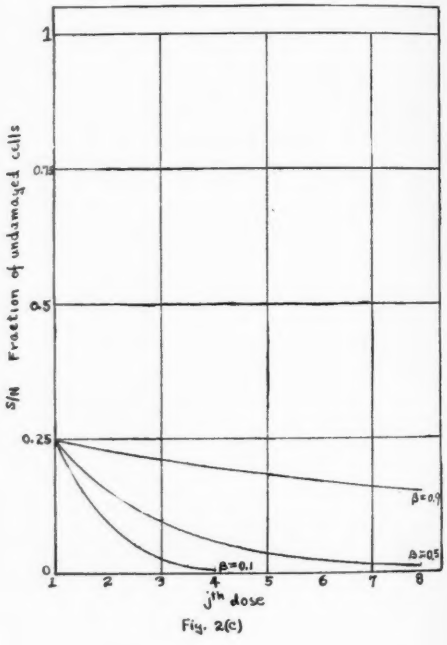


Fig. 2(c)

plotted from Equation (7). According to Crowther, the number of cells damaged by n hits in a dose q is

$$d = Ne^{-\lambda q}\frac{\lambda^n q^n}{n!} \tag{9}$$

For convenience we will write Equations (7) and (8) as

$$S = N\phi(n) \quad \text{and} \quad d = N\alpha(n),$$

respectively. It may be assumed that the sum of the fraction of undamaged cells plus the fraction of damaged cells is unity. This places a unique condition on n. Theoretically it would be impossible to write

$$\frac{d}{N} + \frac{S}{N} = \phi(n) + \alpha(n) = 1.$$

Yet for our purposes this condition will suffice in a first approximation. We may now calculate the number of cells un-

damaged after successive doses. In the first approximation we make the following assumptions:

(1) The cells are not dividing rapidly. This assumption is consistent with the hypothesis that N remains constant.

(3) A fraction of the cells damaged will recover in the interval between doses. Thus if d cells are damaged, then βd cells will recover, where $0 < \beta < 1$. The fraction β will, of course, depend on the dose D and on the time interval T. It

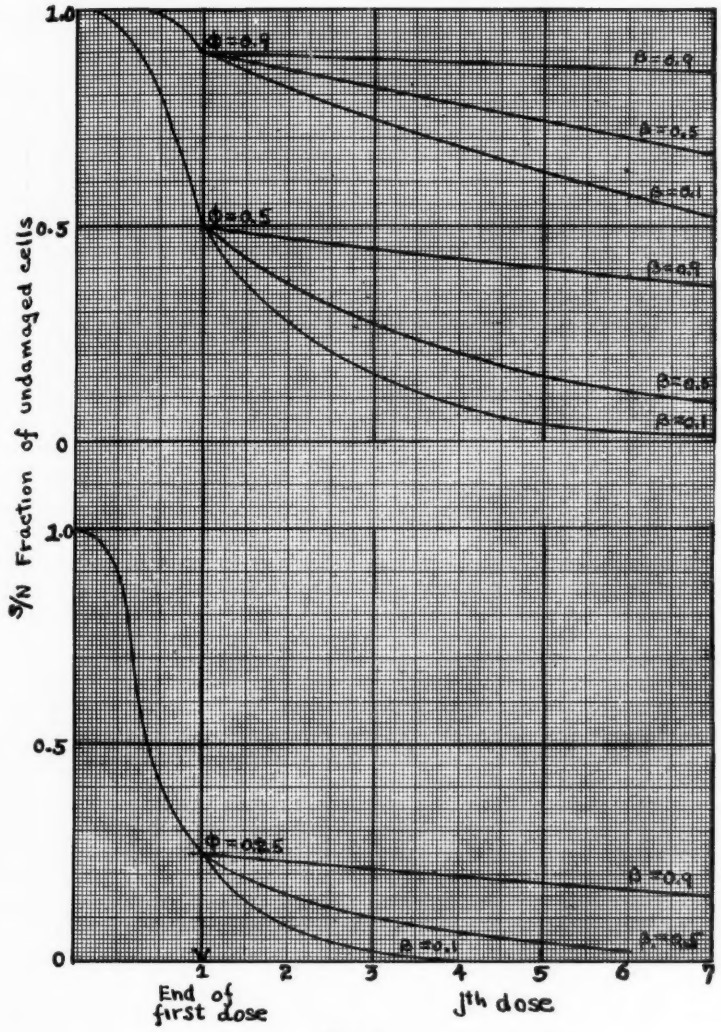


Fig. 3

(2) The probability, λ, of a cell being hit by unit dose is constant throughout the experiment. This is possible since N remains constant and the geometry of the tissue remains unchanged.

may vary linearly or exponentially with T.

Counting the "undamaged" cells, we get for the number of them after the first dose

$$S_1 = N\phi(n)$$

and after the second dose

$$(\rho = (1 - \beta) \text{ and } \omega = 1 - \alpha(n)\rho)$$

$$S_2 = [N - d_1 + \beta d_1]\phi(n) = [N - d_1\rho]\phi(n)$$
$$= \{N - N\alpha(n)\rho\}\phi(n) = N\omega\phi(n)$$

where d_1 is the number of cells killed by the first dose and βd_1 is the number that recovered in the interval between the first and second doses. For the number of cells undamaged after subsequent intervals we have: letting $\phi(n) = \phi$ and $\alpha(n) = \alpha$,

$$S_3 = \{N - d_2 + \beta d_2\}\phi = \{N - N\alpha\rho - N\rho\alpha\omega\}\phi$$

$$S_j = \{N - d_{j-1}\}\phi = \left\{N - N\alpha\rho\frac{1 - \omega^{j-1}}{1 - \omega}\right\}\phi$$

or:

$$\frac{S_j}{N} = \left\{1 - \alpha\rho\frac{1 - \omega^{j-1}}{1 - \omega}\right\}\phi = (1 - \alpha\rho)^{j-1}\phi$$

(for j doses; j = an integer) (10)

The function $S/N = \phi(n)$ has been discussed from the mathematical point of view by Condon and Terrill (15); it can be evaluated from tables of the incomplete gamma function (11). When plotted against q for various values of n and λ, it gives Galton's famous ogival curves as shown in Figure 1. If n is large, there are few damaged cells for a small dose. If n is small, damage sets in very soon after exposure to radiation. In Figure 2 are

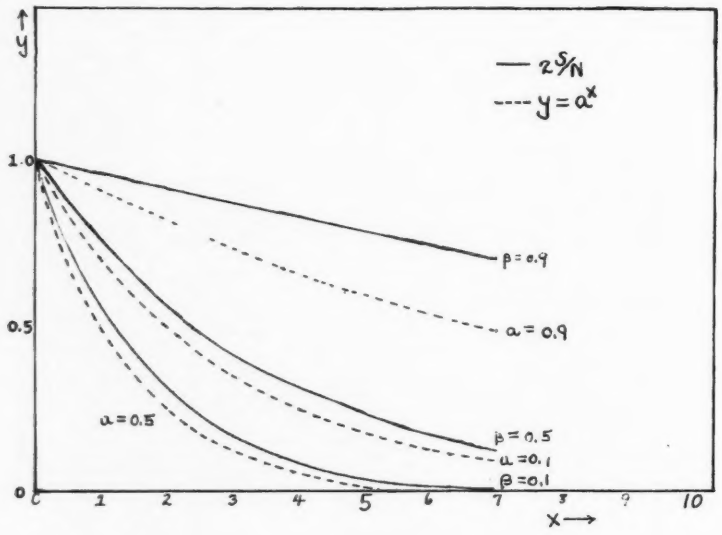


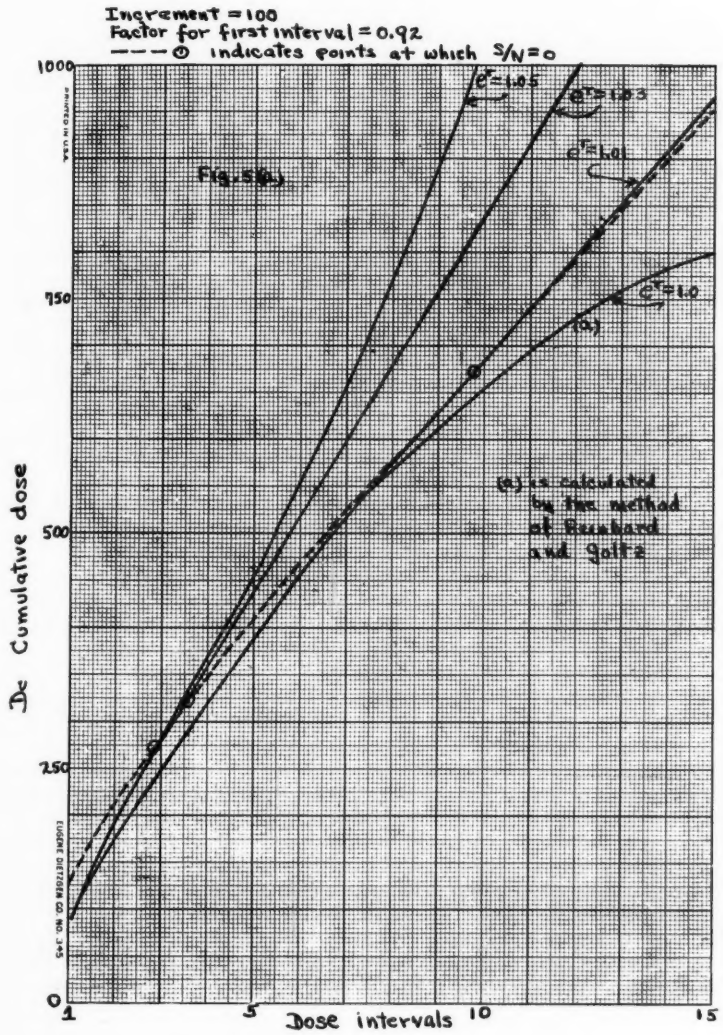
Fig. 4

shown values and the general trend of S/N for various values of $\phi(n)$, the fraction of undamaged cells, and β, the fraction of cells that recover in the interval. The dotted lines indicate the probable course of S/N, while the solid line indicates the apparent course as determined just after each increment of dose is administered. Figure 3 is a composite of Figures 1 and 2, and is an attempt to show the course of S/N beginning with the first dose.

Inspection of Figure 3 shows how widely the reaction of our idealized tissue may vary according to the different modes of irradiation it undergoes. The value of the function $\phi(n)$ is determined by the magnitude of the dose q: it is also determined by n, which in turn would be a function of the quantum magnitude hv, where h is Planck's constant and v is the frequency of the incident radiation. The

experiments of Braun and Holthusen (7) on *Ascaris* eggs seem to indicate that the magnitude of the quantum has no influence on the rate of mortality. They employed radiations in the range of wave lengths from ultra-violet to x-rays of 162 kv., 1 mm. Cu half value. The theoretical calculations of Mayneord (12), however, indicate that an effect due to quantum magnitude is to be expected.

Figure 3 shows that the fraction S/N and, consequently, the recovery factor may vary linearly (approximately) for high values of β, and pronouncedly exponentially for small values of β. This suggests a possible means of calculating the k's in Equations (1), (2), and (3). Figure 4 gives a comparison of the function $y = a^x$ with S/N. The latter has been multiplied by two for convenience of comparison.



Assuming an exponential variation of the recovery factor, we will now calculate

the cumulative dose which was given by

$$D_c = D\{k_1k_2k_3 \ldots k_j + k_2k_3 \ldots k_j + k_3k_4 \ldots k_j + \ldots \quad \ldots + k_j\}.$$

Since $k = 1 - c(S/N)$, we have for undamaged tissue $S/N = 1$ and

$$k_0 = l - c = c_1 \qquad (12)$$

cording to the usual processes of summation we get:

$$T\sum_{i=1}^{j} X_i = T[j + \frac{j}{2}(j-1)] = \frac{T}{2}[j^2 + j]$$

and

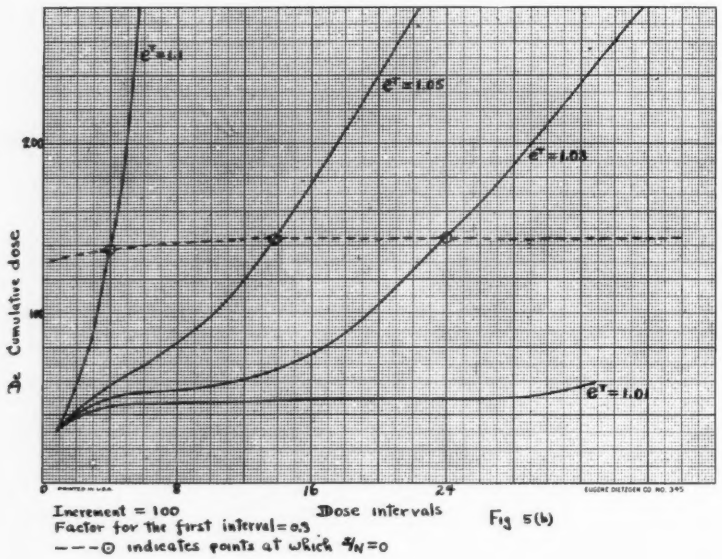


Fig 5(b)

If S/N decreases exponentially, then k increases exponentially, and we write

$$k_0 = c_1e^0 = c_1e^{0T}$$
$$k_1 = c_1e^T, \qquad k_2 = c_1e^{2T}$$
$$k_j = c_1e^{jT}.$$

Note that in order for these factors to have any physical significance, they must fulfill the condition that $c_1e^{jT} \leq 1$. The expression for the cumulative dose becomes; on letting $x = 1, 2, 3, \ldots$, *i.e.*, take on integer values

$$D_c = D\left\{c_1^j e^{T\sum_1^j X} + c_1^{j-1} e^{T\sum_1^{j-1} X} + c_1^{j-2} e^{T\sum_1^{j-2} X} + \ldots \quad \ldots + c_1e^{jT}\right\}.$$

The exponents of e are sums of arithmetic progressions, while the series in the parentheses is a geometrical progression. Ac-

$$D_c = D\left\{c_1^j e^{\frac{T}{2}[j^2+j]} + \ldots \quad \ldots + c_1e^{jT}\right\}.$$

The parenthesis can be shown to be equal to

$$\frac{c_1e^{jT} - c_1^{j+1}e^{jT}c^{jT}}{1 - e^Tc_1} = c_1e^{jT}\frac{1 - (c_1e^T)^j}{1 - c_1e^T}$$

whence

$$D_c = Dc_1e^T\frac{1 - (c_1e^T)^j}{1 - c_1e^T} \quad \ldots\ldots\ldots (13)$$

The constant c_1 can best be interpreted from a consideration of the following idealized experiment: tissue is exposed to radiation of such low intensity that the photo-chemical products are removed from the tissue by the normal processes of elimination rapidly enough to prevent their interfering with the life processes of the cells. Since $k = 1 - c(S/N)$, or

$R = c(S/N)$, c may be termed the recovery factor when $S/N = 1$, or the power of perfectly intact tissue to wash away the effects of radiation. R may be measured in roentgens per unit time. It is evident that c will vary with the *rate* of absorption of energy in the tissue. Once c is determined, c_1 can be obtained from the relationship $c_1 = 1 - c$.

Figures 5, *a*, *b*, and *c*, show the course of the cumulative dose, for an incremental dose of $D = 100$ units. Figure 5-*a* assumes that the recovery factor for the first interval is 8 per cent as used by Reinhard and Goltz (1), or that 92 per cent of the first dose remains in the tissue after the first interval. This initial value is then varied exponentially according to the equation $k = 0.92e^{Ti}$ where $i = 1, 2, 3, \ldots j$; and T has values such that $e^T = 1.01$, $e^T = 1.03$, $e^T = 1.05 \ldots$ etc.

These curves show the cumulative dose just before each increment is given. Figure 5-*b* assumes an initial recovery factor of 70 per cent as suggested by the work of Duffy, Arneson, and Voke (13), who found that skin recovers 69 per cent in 24 hours and 76 per cent in 48 hours for x-radiation of 0.16 Å. effective wave length. Figure 5-*c* assumes a value intermediate between 8 and 70 per cent, namely, 40 per cent for the factor in the first interval. The circles in each diagram indicate approximately where the ratio S/N becomes zero, that is, the point at which all cells are damaged by n hits. Beyond this point the cumulative dose is merely the total dose since the tissue is no longer able to recover from radiation effects.

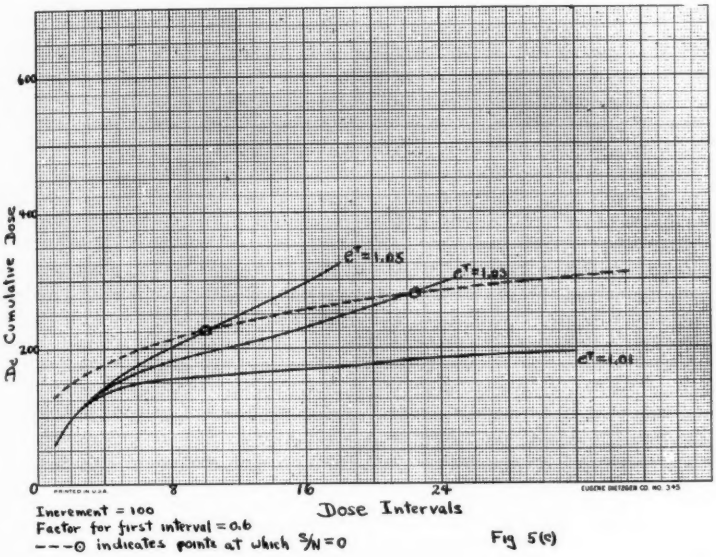


Fig 5(c)

The phrase "damaged by n hits" does not necessarily mean that the cells are dead or have actually suffered mechanical damage. To use the apt word first suggested by Crowther, the cells are in different "states." And the fraction S/N may be taken as an index of the state in which our idealized tissue is after the irradiation. The existence of such states is indicated roughly by Flaskamp (14), who gives a graph indicating reversible and irreversible states in terms of the skin erythemal dose: there exist certain critical doses, which, if exceeded, put the skin in an irreversible state which leads to permanent injury. Crowther found that the organism, *Colpidium colpoda*, was able to recover from roentgen radiation in three hours on the average for moderate doses, but that if a

certain value were exceeded the organism went gradually to a premature death.

The curves of Figures 5, *a*, *b*, and *c*, were calculated for 100 units of incremental dose for convenience of presentation. The cumulative dose for any increment can be obtained by multiplying the ordinates of the 100 unit curves by the proper multiple of 100. Thus for 500 units incremental dose, the ordinates of the 100 unit curves are multiplied by 5. But, since the dose is larger, the dotted lines which give the points at which $S/N = 0$ will be changed. That the point at which $S/N = 0$ varies with the dose and recovery factor is shown by a comparison of Figures 5-*a* and 5-*b*. The slope of the dotted line is much steeper in the former. A noteworthy feature of Figure 5-*b* is that the dotted line is almost straight ($D_c \doteqdot 144$), indicating that the same cumulative dose is required to produce the same biologic effect, but that the effect is attained after different times according as the recovery factor varies. When the recovery factor varies according to the equation $k = 0.297e^{iT}$ where $i = 1, 2, 3, \ldots j$, the cumulative dose never crosses the line where $S/N = 0$. This would correspond to an irradiation which did not produce the biologic effect defined by $S/N = 0$.

In the foregoing calculations we have dealt with an idealized layer of tissue one cell thick. In practical cases one deals with three dimensional masses of tissue. The dose, g, for the different layers of tissue will be different on account of absorption of the radiation. Assuming the simple exponential law for the decrease of intensity with depth with appropriate scattering correction, and assuming a variable sensitivity throughout the volume of the tissue, it becomes a matter of integration over the volume to find the number of cells damaged by a given dose at the surface. Such integrations could be simplified by carrying them out over simple geometrical shapes such as the sphere, pyramid, cylinder, and prism.

We have assumed that the recovery factor is directly proportional to the fraction of the cells damaged, $R = c(S/N)$. In general we should write $R = f(S/N)$. R may actually be proportional to some power of S/N, or may vary exponentially with S/N. We have taken the direct proportionality for simplicity of demonstration, hoping that the theory rather than any specific values may be of some interest. Moreover, we have not yet established a relationship between the ratio S/N and a standard biologic reaction, such as, for example, the skin erythema. Experimental and clinical data will have to determine the nature of the relationship $R = f(S/N)$.

Equation (7) gives the number of cells that have not yet been hit n times; it is derived on the basis that the N cells are independent of one another. Yet it is known that the cells of a tissue are not independent of one another. An added refinement in calculations with an equation of this type would consist in the use of the concept of "virtual hits." A given cell is affected by the actual hits on the other cells. These virtual hits would not necessarily have an immediate effect, but would exert their influence after a time interval depending on how quickly the disturbance is propagated through the tissue. The existence of these virtual effects would be taken care of by assuming a different value of n for each dose throughout the treatment.

If S_i denotes the number of cells undamaged after the i^{th} dose, and $\phi_i(n)$ denotes the probability distribution function which takes into account the virtual hits as well as the actual hits per cell, and β_i and d_i are the fraction of the cells recovering in the i^{th} interval and the number that are damaged by the i^{th} dose, respectively, our equations for the number of undamaged cells after each dose become

$$\begin{aligned} S_1 &= N\phi_1(n) \\ S_2 &= \{N - d_1 + \beta_1 d_1\}\phi_2(n) \\ S_3 &= \{N - d_2 + \beta_2 d_2\}\phi_2(n) \\ &\cdots \\ S_j &= \{N - d_{j-1} + \beta_{j-1} d_{j-1}\}\phi_j(n). \end{aligned}$$

Calculations based on the method of varying n have yet to be made.

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SOME LAWSUITS I HAVE MET AND SOME OF THE LESSONS TO BE LEARNED FROM THEM

(Fourth Installment)

By I. S. TROSTLER, M.D., F.A.C.R., F.A.C.P., *Chicago*

A CANADIAN (PRURITUS) SUIT

A RADIOLOGIST in eastern Canada wrote me, "May I presume on your good nature in stating a case and asking advice?

"I am threatened with a suit as a result of a burn. The patient had pruritus and was given a dosage which appears all right, *viz.*:

"March 3, 1931, 3 ma., 3 inch spark gap (kilovolt measurement), 10 inch distance, no filter, for five minutes' time.

"March 21, in 18 days, she had another treatment for two minutes with the same factors.

"April 13, 23 days later, I saw her and described in my notes an erythema which I thought was satisfactory.

"July 2, 3 months later, another physician saw her with an ulcer on the left buttock.

"Sept. 2, 1932, to-day, several physicians (my friends) state that the healed appearance of the region about the anus is that of an x-ray burn.

"The thing that suggests itself to me is that she received some further x-ray treatment elsewhere; but that is only conjecture and incapable of proof by me.

"Would it be too much trouble for you to suggest possible lines of defense?

"Could you give me your opinion on the question of idiosyncrasy? And quote me authorities?

"Could you suggest any especial medical x-ray authorities in the eastern United States, who might be willing to appear as expert witnesses? I feel sure that I would have the absolute support from Canadian x-ray men. The opposition is getting a Dr. K—— T——, from Boston.

"The main reason I am writing you is that I know of your great interest in the medico-legal end of our specialty, and again, in Canada there is very little precedent, either legal or medical, upon which we can call.

"Thanking you, Doctor, in anticipation," etc.

I replied: "Replying to yours of recent date, will say that if your laws regarding medical malpractice are at all like ours, you should have little or no difficulty in getting a non-suit, and the case taken from the jury by the presiding judge, because you can immediately show that your treatment was not in any sense or possible way malpractice or wrong in any way.

"If you have 'X-Rays and Radium in the Treatment of Diseases of the Skin," by George M. MacKee, second edition, 1927, and will refer to page 259, you will find a table of dosage for 3 inch spark gap technic for 8, 10, and 12 inches and for 2 and 3 milliamperes.

"The technic you used—10 inch distance, 3 inch s.g., 3 ma., for 5 minutes—will produce just short of 43 per cent of an E.S.D., while your second application administered 18 days later, of 2 minutes at the same technic, will produce exactly 17 per cent of an E.S.D., or a total of just short of 60 per cent of an E.S.D. over a period of 18 days. *If that produced even an erythema*, either your measurements are wrong or the patient had a marked idiosyncrasy.

"I do not know how your laws are; but if there is an opportunity to introduce MacKee's book in evidence, and I know how I could do that, *that table*, even if they do not allow the introduction of books, there should be no trouble in getting a directed verdict, even if the case was allowed to go to the jury.

"You ask me for the names of competent experts in New England. The nearest one is a woman physician, whom I am sure can put it over for you. [Here I cited the names and addresses of six physicians in

the area for which he asked.] All of these can qualify as experts, and will, I know, be glad and willing to testify that the treatment as indicated to me was conservative, well within safe limits, etc.

"That Dr. K. T., of B——, was born in 1899, graduated from . . . Medical College in 1921, was licensed to practice in M—— in 1921, poses as a dermatologist and is so listed in the A. M. A. Directory. He is a Fellow of the American Medical Association and his address is . . . I know nothing about him; but if you want me to, I can find out something from our Counsellor for M——. You might write him yourself, sending a postage paid envelope.

"Now as regards idiosyncrasy. There are quite a number of decisions from high courts upon it. Among some of these are those listed in MacKee's book, page 1267 of the second edition. You should have this book.

"I will be glad if what I have written is of some help to you, and if I can help further, be free to call upon me; but please pay the postage fully. I had to pay postage due on your last one."

I heard no more about this case until I recently wrote to ask about the outcome, and received the following:

"The case came to trial and a judgment was rendered for the plaintiff, based entirely on the judge's impression that a definite warning of possible danger should have been given to the patient. This judgment was appealed and here an interesting incident took place, namely, that the court stenographer died before the notes were transcribed. The appeal judges, therefore, gave as their opinion that a new trial was the fairest in this particular case.

"At the new trial, before another judge, the same witnesses and evidence, the case was dismissed, the judge basing his opinion on the following fact—that the claim was outlawed by the local statute of limitations. However, he went fully into every claim of negligence and gave the defendant 'a clean bill of health' on every point, with the added rider that the plaintiff and her witnesses showed a woeful lack of veracity in both their claims and evidence.

"Dr. T., of B——, did testify, but his evidence was rather shaken and discounted by other evidence in rebuttal by my witnesses, particularly *re* his qualifications; as it turned out that he was a graduate of a very poor school and his other qualifications were of like character.

"We did not use any of the witnesses recommended by you. On the other hand, one of my competitors here was one of my best witnesses. Dr. P——, of M——, was my other radiological expert witness.

"The case was reviewed in the March, 1935, issue of the Canadian Medical Journal, page 329, the synopsis being made by a lawyer for the Canadian Medical Journal."

Malpractice suits are not nearly so frequent in Canada as they are in the United States, and our Canadian brethren are to be congratulated upon winning this one, as one such case lost would be a signal to the legal profession that there was a likelihood of more of the same sort to be won. One such case defeated is better than any number settled either out of court or tried and lost.

NOT NECESSARY TO INTRODUCE ROENTGENOGRAMS?

A radiologist in the Southwest wrote me: "Some time during the past few months, I recall seeing a court ruling, in which it held that it is not necessary to introduce radiographs, but the roentgenologist's expert testimony is sufficient. I have an occasion to request that ruling through my attorney in a case in which I expect to appear very soon and want to give him the reference, but have not located it. Knowing your special interest in the medico-legal aspects of radiology, I wonder if you recall the ruling, and if you can tell me where it can be found."

I replied as follows: "Replying to yours of the 24th, will say that I do not remember having seen the recent case decision you mention; but having a nearly completed paper on this and allied subjects[1] on my desk, I can easily give you several refer-

ences which I am sure will serve to put you in the position you apparently desire to be.

"Interpretation of a roentgenogram which was lost and, of course, not in evidence, was admitted in Speilmeyer *vs.* Hiertz Metal Co., 272 S.W.R., 1068.

"A similar decision was rendered in a case in which the witness was a chiropractor, in Bachman *vs.* Quincy and K.C. Ry. Co., 274 S.W.R., 761 (a Missouri case).

"Quinn *vs.* Flesher, 102 S.E.R., 300, and Marion *vs.* Coon Construction Co., 110 N.E.R., 444, also point that way. The last-named case is a very important one and well worth reading.

"On 'the other side of the fence,' Lefebure *vs.* Western Coal & Mining Co., 285 Pac. R., 450, specifically states that 'An expert witness may not testify as to what a roentgenogram shows, when the same is obtainable and not in evidence.' This is an interesting case.

"If your attorney or you come upon the case you had in mind when you wrote me, *or any others along the same line*, I wish that you would give me the citations for my files. I am very much interested in that particular subject."

Although the above letter was written nearly two years ago, I have not had a line from my Southwestern friend. Like many others, perhaps he is too busy to write, *except when he wants something*. I sometimes wonder whether it is worth while for a fellow to acquire a reputation on some subject. I am asked questions like the foregoing, from four to six times every month, and the time spent in digging out the answers amounts up considerably. If the ones benefited would even as much as thank me for what I do, I would not care; but some of them seem to think that it is my duty to do these things. Perhaps it is, but . . . Well, anyway, I suppose I will keep on doing it as long as I live. I have always been a sort of "ask George" sort of fellow, and probably would miss it if I was not asked.

[1] The Legal Aspects of the Identification and Interpretation of Roentgenograms. I. S. Trostler. Am. Jour. Roentgenol. and Rad. Ther., November, 1934, **32**, 680–693. Reprints furnished at 12 cents each.

A SUIT INVOLVING A RADIUM TREATMENT

Ten years ago I was consulted relative to the defense of a malpractice suit, wherein an Illinois physician in treating a pigmented hairy nevus on the elbow of a three-year-old child, applied 115 milligrams of radium screened with 1 mm. of silver(?), 1 mm. of copper, 2 mm. of rubber and five or six thicknesses of gauze for two hours (230 milligram-hours), according to the records of the defendant physician.

It was alleged that a violent reaction followed, with a marked arthritic involvement of the child's elbow and considerable permanent disability and deformity as the end-result.

I advised stalling the trial along as long as possible, by means of the various methods so well known to the legal profession, with the idea that if any injury had been done, the natural recovery of tissue and particularly in joints in children, would at least minimize the degree of involvement, and thereby lessen the likelihood of large damages being assessed.

The parents of the child had brought suit for $25,000. After stalling along for two years, it appears that the defendant's legal counsel were unable to secure any more continuances, and the suit finally settled for $5,000, which, as far as I could learn, was more than the real damages were worth, as I was informed that the child's elbow was coming rapidly back to nearly—if not quite—normal.

READING OR QUOTING FROM MEDICAL BOOKS, AS TESTIMONY

A far Western radiologist who had practised in Chicago became involved in a suit wherein certain medical books were referred to while he was on the witness stand. He wrote asking me about "testimony quoted by attorneys from the author of books, which was ruled out by some Superior Court, as being hearsay testimony and not admissible."

I wrote him in part as follows: "I am not sure that I understand exactly what you want to know, or, rather, what you want

me to give my opinion upon, when you say 'testimony quoted by attorneys from the author, was ruled out,' etc.

"If you mean the reading of medical books to the jury, as evidence for the purpose of influencing that jury, I would say that there are plenty of references to indicate that 'the contents of scientific books cannot be read to a jury for the purpose of establishing the facts or establishing the deductions stated in them.' (See references at end hereof.)

"Another aspect of this situation is that, 'It is well established (according to several New York State high courts), that a party calling an expert witness cannot read from medical works on inductive science and ask his expert if he agrees with the statement of the author, or if it accords with his experience.' (See references 6 and 7.)

"But to all this, there are notable exceptions and *by very good and highly respected authorities.* (See references 3 and 4.)

"On the other hand, when it comes to cross-examination, an entirely different rule applies. During cross-examination, the qualification of an expert witness may be established 'in any way which will enable the jury, who are to pass upon the weight to be given to his testimony, to judge intelligently about it.' (See reference 1.) It is permitted and considered permissible in some States in this country to call the attention of expert witnesses, during cross-examination, to medical books and to ask them if they agree with the authors. This is, of course, not done for the purpose of trying to make the contents of the books into evidence, but is usually done for the purpose of belittling the witness and of minimizing or reducing the weight of his testimony. The degree and extent to which attorneys may go in such questioning depend much (if not entirely) upon the presiding judge.

"I note what you say about my feeble efforts to bring medico-legal information to the readers of RADIOLOGY. I have had many complimentary letters about it, and know my efforts are appreciated. We do not know enough about this important subject.

"While on the subject of the introduction of medical books and questioning expert witnesses, in cross-examination, to belittle and reduce the weight of their testimony, I am reminded of an instance in which this was being 'worked' on me. While holding a big, formidable looking book in his hand, a very prominent attorney asked me if I agreed with what the author said about the subject of my testimony. Being on my guard against some trick or subterfuge, I said I was unable to state, until I saw the book more closely. The lawyer pompously handed me the book, and, upon opening it at the title page, I discovered that it was more than twenty years old. Handing the book back to the attorney, I said that practically no medical book as old as the one he had in his hand was considered to be worth the paper it was printed upon. He hurriedly laid the book upon his table and started another line of questioning. In re-direct examination, I was asked about how old the book just mentioned was, and when I said more than twenty years, I could see by the expression on the faces of the jury that my testimony 'went over' with them. I gave out the impression that the big attorney was trying to trap me and that I not only could not be trapped, but trapped him. Juries enjoy such things as well as any, if they have the I.Q. to appreciate them.

"If this is not the information you want, make it just a little clearer and I will see if I can furnish it."

A CHICAGOAN'S ACNE CASE

The attorney for an insurance company sent a young woman to see me, whose attorney had threatened suit against a young Chicago physician for malpractice in the application of the roentgen rays in the treatment of acne on her face.

The claimant had had acne for about five years and had received twelve applications of the x-rays: the first four were 3 minutes, at 15 inches F.S.D., and the last eight were 12 minutes with the tube at 10 inches

F.S.D., all unfiltered and little known of the voltage or milliamperage. The fee paid for the course was $250.00.

I reported as follows: "*In re* L—— N—— *vs.* Dr. N—— T——: Pursuant to your instructions, I have examined this prospective plaintiff and hereby report.

"I find that there is at this time a condition known as telangiectasis, accompanied with beginning skin atrophy on both cheeks—involving the mesial areas of both cheeks, the upper lip, the lower lip, the lower edges of both nasal alæ, the nasal septum (externally), and the anterior aspect of the chin. There is also some considerable induration and irregular scarring in these areas, most of which are on the chin, and cheeks. The vermillion surfaces of both upper and lower lips are also involved in this change.

"While some—an indeterminate part—of the roughness and induration in the involved areas may be due to the forerunning acne, for which this girl was treated, there is absolutely no question that by far the greater portion of it was produced by overdosage of the roentgen rays.

"The prognosis in this case is in my opinion about as follows: The telangiectasis will become more marked and the skin atrophy, which is now only in the beginning, will progress and become more evident in a year or two. *There is definite danger that this girl may develop malignant disease in the involved area.*

"She informed me that the last roentgen-ray treatment was administered in September, 1931, and that she was not twenty-one years old until January 29, 1932, and I have no doubt that this is the truth. In other words, she was a minor or legal infant until the last-named date (four months ago). This may have some bearing on your disposition of the case.

"There is no question but that the treatment administered to this patient was the grossest of error and that she has been irreparably damaged. Likewise, there is no question that her appearance at this time is decidedly better than it will be in a year or two from now.

"I believe that I have given you the information you asked for and will need in disposing of this case. If more is desired, or I can be of further service regarding same, command me."

The insurance company paid this patient $2,000 and, if I am any judge of the matter, will say that they got off cheaply. The young Doctor was not paying the additional fee rate for the use of the x-rays at that time. I understand that he is not now treating by means of the x-rays. His father, also a physician, sent me a patient for roentgen therapy about a year later.

A TECHNICIAN TREATED A PHYSICIAN'S PRURITUS

A technician employed in an x-ray and clinical Laboratory conducted by a physician was one of the co-defendants with his employer, because of his (the technician's) x-ray treatment of a pruritus upon the person of another physician. He wrote me: "Pursuant to our conversation of January 24, I am enclosing information relative to the x-ray dermatitis case in question. To the best of my knowledge, conditions in this case are as follows:

"On October 26, 1923, the Doctor (plaintiff) came to me requesting that I give him the following exposure; 5 minutes, 5 inch spark gap, 5 ma., 12 inch target skin distance and no filter. This prescription was carried out as carefully as I was able to measure same. A total of eleven such treatments was given, as indicated on treatment slip (the ten applications were given in exactly eight months at about equally divided intervals).

"During this series of treatments no erythema was in evidence although careful examination was made prior to each treatment. No treatments were given from June 25, 1924, until March 21, 1929, during which time frequent local drug medication was used by the Doctor. When he returned for treatment on the last-named date, I called his attention to a marked redness about the anus, which appeared as though it had been severely scratched.

"Before resuming x-ray treatment, I

showed the Doctor a letter which I had received from a radiologist, relative to a similar case, from which I quote the following: 'Mr. . . . came to me June 16, 1928, with a history of pruritus of five years' standing. I gave him 75 kv., 1.5 minutes, 5 ma., at 12 inch distance and no filter. This same dose was repeated on the 23rd and on July 6th. I usually treat these cases every week or ten days and continue the treatments several times after all the symptoms have disappeared. I felt at the time that I was treating Mr. . . . that he should really have several more treatments, but he did not feel inclined to have them. These cases are usually very stubborn,' etc.

"After reading this letter the Doctor (plaintiff) asked that I give the same treatment to him. Four such treatments were given in this series and each time the Doctor came in, I called his attention to the reddened condition. It is interesting to note that our stories do not agree relative to the time that this redness appeared. I maintain that it was there before the last four treatments in 1929, while the Doctor insists that they did not know of its existance until after the last four treatments were given. He states that he visited Dr. O . . . . at that time and was informed that he had a telangiectasis caused by too much x-ray. My recollection is that he visited Dr. O. . . . after we had completed our treatments in 1924.

"All went quietly and we had dismissed the matter from our minds when, on Sept. 18, 1931, the Doctor came to me, stating that he had to have something done and asking me to let bygones be bygones and that he was willing to take the responsibility of future treatment, and to say that Dr. O. . . . was mistaken in his diagnosis.

"After giving two more treatments I again called the patient's attention to the unsatisfactory condition and suggested that he consult some specialist, as I was not satisfied about continuing to give him x-ray treatments. He (plaintiff) suggested that lack of results was due to insufficient exposure, so we decided to extend the time in the last four treatments. This was under protest by me, but insisted upon by the Doctor, and was supplemented by various medication, sitz baths, etc.

"It is hardly necessary for me to say that had I followed my better sense of judgment in this case instead of the Doctor's orders, no treatments would have been given after the first eleven. I might add that the same technic as used in the last four treatments has been used on other cases with good results and no ill effects, which, of course is not much consolation as far as the Doctor's case is concerned.

"This is a long story and I assure you that we are always glad to learn of a new and better technic in connection with x-ray work."

I replied immediately as follows: "I am sorry to say that I most strenuously disapprove of the treatment you administered and do not believe that you will be able to find a roentgenologist of experience and reputation, or one of any standing in radiology at all, who will be willing to go on the witness stand and testify that it was good therapy, or properly administered x-ray treatment.

"Your repeated application of unfiltered radiation in this case is contrary to what I personally have for many years been harping against, and while quite a number of dermatologists still apply a considerable number of unfiltered treatments, none of them would dare apply as much at each time as you did. There is no question in my mind but that you administered too much radiation during 1922 and 1924, and that, while the amount in 1929 and 1931 were probably not enough to damage a normal skin, it was probably enough to seriously injure the already insulted skin.

"So, reasoning from the above premises, as I see things, the only useful defense that you can have will be that the patient, a graduate and licensed physician, prescribed the treatment, and all you did was to fill his prescription. In other words, the patient, acting as his own physician, assumed the relation of principal and you assumed the position as his agent. If that can be presented to the attorneys, or to the court

and jury, and the fact that a principal is always responsible for the acts of his agent, you and your employer may be released from responsibility. Personally I think that this can be done, fortunately for you and Dr. G.

"While I have no right to scold you, much as I think you deserve it, I cannot help saying that in my opinion, you should *never* administer any treatment until some physician gives you a prescription or formula for the case or patient to be treated. If you do that, you will be on the safe side, and whoever writes the prescription will be responsible."

I heard nothing further for nearly four years, when, upon inquiry from the employer (physician defendant), he wrote: "The suit you mention in which a fellow physician was suing me for damages for the small sum of $50,000 was withdrawn by him. The only agreement that I made was that I would not sue him for damages. No money was paid. As the case did not go to court, I could not tell you just exactly what the defense would have been but it would probably have been on the basis of master and servant—the patient in this case being the master and my technician the servant, as you outlined. Thanking you for your kindly interest and advice to my technician," etc.

This suit was the third of a similar kind, in which a physician insisted upon having treatment which ultimately damaged and injured him, and wherein I was able by the presentation of the principal and agent defense, to secure the withdrawal of or prevent the filing of suits against the agent by the principal. Admitting that this may not be a nice thing to do, we must also realize that malpractice suits are not nice, and, from my way of thinking, practically any means or method of defeating them is and should be considered justifiable. I repeat, *malpractice suits are not nice*, at any time or in any sense, nor are they to be defeated by niceties. They require hard-boiled tactics, and when one has been in the fight against them as long as has the writer, one cannot help becoming pretty obdurate and repellent regarding them.

FORTY-EIGHT UNFILTERED ROENTGEN-RAY TREATMENTS

A middle-aged man, complaining of a chronic eczema on the backs of both his hands, received the following from a Chicago dermatologist according to the dermatologist's records: 7 treatments from a gas tube in 1919; 8 treatments in 1923, each of which was reckoned to be one-fourth skin unit according to the formula $\frac{\text{ma.} \times \text{min.} \times \text{S.G.}}{\text{distance}} = \text{dosage}$. In 1924 the patient received 30 applications of one-fourth skin unit each and in 1925 three more of the same intensity. *All of these were unfiltered.*

A few months following the last application (in 1925), this patient consulted a very prominent dermatologist, and was informed that he had received "x-ray burns." Somewhat later he visited a well-known clinic, where again he was informed that he had "x-ray burns." Shortly thereafter he brought suit against the dermatologist who had administered the treatment, and in 1928 I was consulted relative to helping outline a defense and assisting in the trial of the case.

After I had learned the particulars, as related above, I informed all concerned in the affair that I could neither testify that the treatment was correct and proper, nor could I even suggest any adequate or useful defense for the case. I told then that "*it just ain't.*"

The plaintiff's attorneys were unable to induce any physicians to testify for them, but managed to induce an apparatus salesman from a neighboring State to testify. They paid him $100 for his services but he repented his action for a long time, and told me afterward that he would not do it again for $10,000.

The trial resulted in a verdict for the plaintiff, which was later settled for $3,000.

X-RAY THERAPY—AND HOW!

In a recent suit for malpractice in Chicago, the physician defendant owned x-ray apparatus and employed his brother as

technician, to operate the same. The physician has no knowledge whatsoever regarding roentgentherapy and his brother—the technician—knows only how to throw the switches. No records of voltage, time, F.S.D., or milliamperage were kept and they knew nothing about filters—in fact, they had none. In addition to their not having records, even the dates of the application of the radiation treatments were indefinite.

A large number of applications of unfiltered radiation were applied to the backs of both hands of the plaintiff, in the treatment of a chronic eczema, with the result that much damage was done.

I was consulted by another physician and the defendant's attorney regarding my appearance as an expert witness and the planning of a defense, and apparently to their great surprise, I declined to testify or offer them any plans leading toward a successful defense.

This was one of the worst instances of the misuse of the roentgen rays that I have ever seen, and I could not see my way clear to mix into it in any way. It was one of the far too numerous reasons why many otherwise intelligent people fear to have x-ray therapy applied, when such therapy is needed and most applicable for their trouble.

Investigation proved to me that it was merely an example of cupidity on the part of the pair involved in the application of the treatment. They had heard, or had been told by an apparatus salesman, that much money was to be made by owning x-ray apparatus. They had bought same and used it whenever and wherever the opportunity presented itself. In fact, they made the opportunities.

The case was settled out of court for $4,000, which was cheap enough, at that price.

ANOTHER ECZEMA CASE

Eczematous skins appear to suffer more from roentgen-ray injuries than do any others. At least your relater hears about more such instances—wherein patients who have eczema have injuries and the physicians who treat them get into court because of them—than of any other group of skin diseases.

Eczema is a very common skin disease, and it may be because of this and that our radiation therapy is known to be beneficial in eczema, which accounts for the number of cases of treatment of eczema that get into court.

In 1930 I received the following letter: "I have been reading some of your articles on expert testimony and medico-legal cases from time to time, therefore I am writing you regarding a suit which I have pending. The case is one of alleged x-ray burn following a course of treatments given by me. The secondary changes apparently did not develop until fully a year and a half following the last treatment and I have been advised that keratoses with a few areas of malignant degeneration have developed over the area treated. There is, however, another factor that entered into the case, namely, a self-administration of arsenic in the form of Fowler's solution over a long period without my knowledge, so you see there is an opportunity for a difference in opinion and possibly expert testimony.

"Along about last January at one of our medical meetings at S . . . . C . . . . I heard a Dr. P. . . ., of Chicago, give a talk on various types of neuritis and one of the types which he very carefully elucidated was that of arsenical neuritis in the hands. The claimant in my case claims loss of strength, etc., in the hands and fingers. I wrote Dr. P. . . . some months ago relative to this matter and he answered me rather unsatisfactorily, although I believe he stated that most of such findings had been worked out by his associate.

"I am wondering if you get a slant on my situation, and if you would care to write me what you think about it. I may also state that the M. . . . P . . . . Co. will defend, but it may be that we should have some expert testimony. Anything you write will be appreciated by me."

My reply was as follows: " . . . in reply, I will say that without more complete infor-

mation than you gave me, I am unable to give an opinion that would be worth the paper it is written upon. You know, Dr. N . . . ., one must know all (or nearly all) about such cases before one can intelligently formulate ideas that are of any real value, and you do not want guesswork, even if I was disposed to guess.

"May I suggest that you write to your insurance carriers asking that I be informed regarding the status of your case, incidently stating that you know me and would like my opinion and advice. They will then send me a complete copy of the files in your case, and, after looking them over, I will give you or them an opinion and such advice as I deem wise in the circumstances. This is the usual procedure in matters of this sort. Has your case been set for trial? If so, when?

"We have three Doctors by the name of P . . . . in Chicago: W. J., a general practitioner; H. L., an otolaryngologist; and L. J., a neuropsychiatrist. I take it that it was L. J. to whom you refer. He is a man of the very highest standing, and one whose statements may be taken as almost the last word on any neuropsychiatric subject.

"I believe that arsenic neuritis is a definite and recognized clinical entity, and in your case may play an important part, if you can get it introduced into the evidence.

"Trusting that the foregoing will be helpful, and assuring you it will be my pleasure and desire to do anything I can toward aiding you in securing a favorable verdict, I am . . ."

The defendant told me at a subsequent meeting of the Radiological Society that the case had been settled for a moderate sum, and disposed of in that way.

ANOTHER ECZEMA CASE[2]

The plaintiff in this suit was a farmer, in a north-central State, who had brought suit against his physician because of alleged malpractice in treating eczema on his hands.

The physician defendant carried no insurance against malpractice suits, so his State medical society fought the suit for him. I was asked, over long distance telephone, to assist in the case and was assured that I would be paid a moderate fee for my services.

The plaintiff was in Chicago and, while here, he made depositions which were to be used in the case. I was asked to attend conferences, consultations, assist the attorneys in the taking of the depositions, and make affidavits in regard to my opinion of the matter, etc.

The case was settled for much less than the plaintiff was suing for. I spent about two hundred dollars worth of time and billed that State society for $50. The services were rendered in 1926 and the bill was paid in 1932. *But it was paid.*

(*To be continued*)

REFERENCES

(1) Egan *vs.* Dry Dock and B. R. R., 12 N. Y. App. Div., 556.
(2) C. J. 923, sec. 1127.
(3) Am. Law Reg., Vol. 54, O. S. N. S. 45, p. 330, No. 6, June, 1906.
(4) Wigmore on Evidence, Vol. 3, p. 2178 (Sec. 1692).
(5) Foggett *vs.* Fisher, 23 N. Y. App. Div., 207; Wharton on Evidence, Sec. 666; Commonwealth *vs.* Wilson, 1 Gray, 337; Washburn *vs.* Cuddihy, 8 Gray, 430; People *vs.* Milard, 53 Mich. R., 63; Epp *vs.* State, 102 Md. R., 539; Harris *vs.* Panama R. R. Co., 3 Bosw., 7; Matter of Mason, 60 Hun., 46.
(6) Matter of Hock, 74 Misc. R., 15, 22; Foggett *vs.* Fischer, 74 Misc. R., 5; Pahl *vs.* Troy City R. R. Co., 81 N. Y. App. Div., 308.

[2] Not so important, but interesting—to me, at least

BRONCHOLITHS AND STONE ASTHMA

By EUGENE P. PENDERGRASS, M.D., *and* ALFRED A. DE LORIMIER,[1] Capt. M.C., U. S. Army, *Philadelphia*

PULMONARY calcifications interested Aristotle. Perhaps, previous to his time, and certainly since his time, they have fascinated the minds of generations of medical investigators (14, 11, 1, 18, 20, and 12). Many of the notations have essentially been that of amazement. Long before biochemical analyses came into usage, postmortem observers marveled at man's ability to form stone in the soft tissues. But even since the advent of biochemistry, our insight into the processes by which calcareous depositions occur has proven very limited. In fact, our knowledge has practically been restricted to mere considerations as to where these concretions are likely to develop and, to some extent, when they are likely to form. The biogenetic nature of their development is not known. Wells tackled this problem (21). He differentiated the terms "calcification" and "ossification," but nevertheless he concluded that "there seem to be no essential differences between the processes involved in normal ossification and in most instances of pathologic calcification; any area of calcification may be changed to true bone in the course of time." Brown's analyses (2) of pulmonary concretions revealed percentages of inorganic constituents very similar to those of true bone.

As far as the lungs are concerned, calcareous deposits may be located, originally, in: (1) the lung periphery, in contact with the pleura; (2) the lung parenchyma, regional to the bronchial tree—either localized to one or a few patches, or diffusely sprinkled throughout; (3) the lumina of the bronchi or bronchioles, and (4) the draining lymphatics. Pathologists are prone to associate, in most of these cases, a preliminary stage of tissue degeneration or tissue necrosis. A tuberculous background is most frequently considered. However, other types of infectious processes, including pneumonia, bronchiectasis, abscesses, and infarctions, might be responsible. In cases in which the concretions are primarily formed in the bronchial lumina, aspirated foreign bodies are frequently the basis. At first, some of these are ray-transparent, later becoming calcified; others, such as rhinoliths, tonsilliths, or dental calculi (15), are sufficiently opaque, by themselves, to produce a shadow, roentgenographically. Silicotic or anthracotic material might also be included in these categories (8). When a sprinkling of calcification is found throughout the lung parenchyma, biochemical bases must be considered. As long ago as 1885, Virchow (20) noted that the lung, the stomach, and the kidney were sites prone to calcifications. Wells (21) attempted to explain these observations on the grounds that such are "places in the body where acids are excreted"—in the lungs "we have bicarbonates giving up CO_2 and passing on in the blood as carbonates; in the stomach, HCl is excreted, and in the kidneys, acid phosphates are excreted by a reaction which leaves basic phosphates and carbonates." When the blood values for calcium and its anions are elevated, deposition of calcium salts might be expected to occur in instances in which there is a relative alkalinity, for there the solubility product is less. Crane (4) noted this type of deposit in the lungs. His case was one of metastates to the skeleton, the primary lesion being in the prostate. The suggestion was made that, due to the displacement of skeletal elements, there had occurred an excess content of the skeletal inorganic elements in the blood stream and, thereby, calcific deposits in the lung tissues. Such precipitations have also

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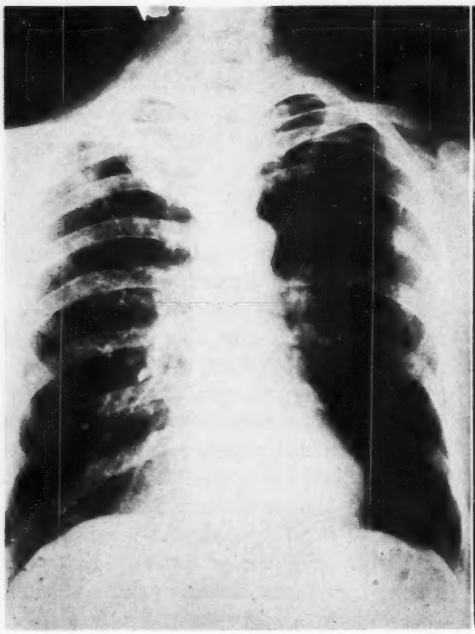

Fig. 1. Case 1. Postero-anterior projection, showing scattered areas of calcification, which are most conspicuous at the right hilum.

Fig. 2. Right anterior oblique position, showing "nest of calcifications," located in the region in which the scarring was noted—bronchoscopically, in the wall of the bronchus.

been found in cases of excessive parathyroid hormone (7), as well as in hypervitaminosis D (17). More discrete calcific lesions, especially in cases in which interval studies reveal an increase in their dimensions, point to the possibility of osteoblastic metastases, *i.e.*, metastases of cells possessing osteoblastic properties. Histologically, these cells are derived from bone-forming tissues (3).

In general, then, when attempting to account for calcific lesions in the lungs, these four possibilities might be considered: (1) secondary calcification after degeneration or necrosis due to infection; (2) primary or secondary calcification of aspirated foreign bodies; (3) precipitation of the osseous inorganic elements, due to excessive concentrations of them in the blood, and (4) metastases of bone-forming cells. Concretions of grossly visible dimensions, produced by any of these four general types of processes, are spoken of as "pneumoliths," "lung calculi," or "lung stones." In the case of aspirated liths, or when parenchymal concretions erode through the walls of the bronchi and become free in the lumina of the bronchi or bronchioles, the term "broncholiths" is applied. These become very irritating to the mucosa. There results the cough reflex, and with it, change in the position of the lith, and further irritation. Excessive mucus is secreted—so much, frequently, that the condition has been described as a "bronchorrhea" or "bronchial colic." Bronchial spasm is likewise characteristic. There develops, therefore, wheezing and expiratory dyspnea—hence the term, "stone asthma." Paroxysms of strenuous and painful cough are the rule. At times, the severity of the cough is so great that patients speak of a "tearing sensation." These cutting pains are even localized to the post-sternal region, or to one or the other lateral portion of the chest. Some of these attacks are climaxed by a powerful expulsive cough, with the production of a stone or of gritty material.

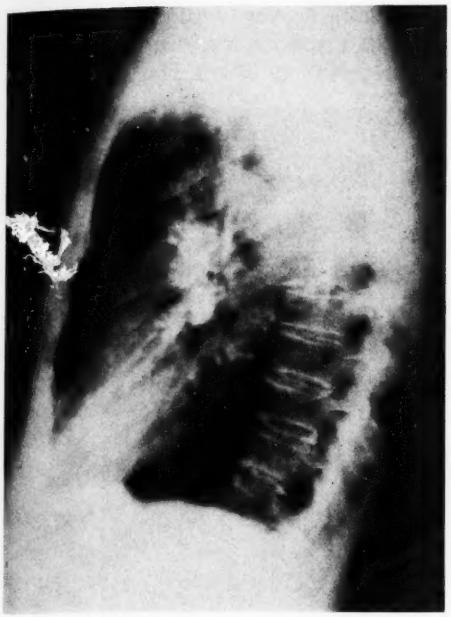

Fig. 3. Lateral projection, localizing the calcifications to the hilum, and revealing a clear retrocardiac region.

Hemoptysis usually occurs when the stone is released, or it may be noted repeatedly during the coughing attacks, and perhaps periodically, over stretches of weeks or months (16 and 19). Such cases are usually branded as active tuberculosis, regardless of repeated negative sputum studies. If secondary infections take place, the essential condition becomes masked by the symptomatology or signs of chronic bronchitis, bronchiectasis, abcesses, or pneumonia.

No doubt many patients cough up small particles of calcareous débris without notice, or with the mere mention of a "grittiness" to their sputum. It is relatively rare that actual "stones" are expectorated. Though it is usually conceded that tuberculosis is likely the most frequent etiologic agent, even in that condition, dislodgment of calcifications is of low incidence. Pritchard (13) noted only two such cases among 7,000 patients under his observation, while Stivelman (19) observed only one actual "stone-spitter" among 5,000 tuberculous patients. Except in those cases of aspiration of foreign bodies, when one lith has been expectorated, more are likely to follow.

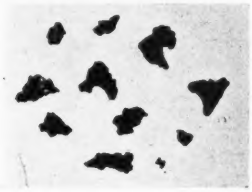

Fig. 4. Photograph of stones collected by patient; expectorated by him during the past six years.

There has been evidence to suggest that repeated sloughings of calcifications take place from the lung parenchyma, or the lymphatics, through the walls of the bronchial tree. It has been suggested that during this process there may occur a fragmentation of bronchial cartilage, and that with subsequent calcification of it, there is developed a nest of stones. Such a process probably accounts for the multiple productions in "chronic stone-spitters." Examples of such would include: Lyter's patient (10), who spat up at least 40 stones over an interval of about six years; Elliott's case (5), from whom 56 concretions were collected within a period of two years; Lloyd's patient (9), credited with 50 stones, and the most spectacular case reported by Boerhaave (1), claiming the expectoration of over 400 calculi, before dying of "phthisis calculosa." Some of these stones have been very large, even to the size capable of almost complete obstruction of the larynx (16).

Though these pathologic conditions and clinical manifestations have been noted for many ages, and even though the symptomatology of stone asthma is relatively rare, it has seemed pertinent to speak again of the condition so that we may be reminded of broncholiths as a possibility in differential diagnosis, and so that procedures might be invoked to quickly alleviate a patient who might otherwise pursue a protracted, painful, and perhaps serious

course of disability. As previously mentioned, in the earlier stages of the process, bronchial spasm is usually a paramount

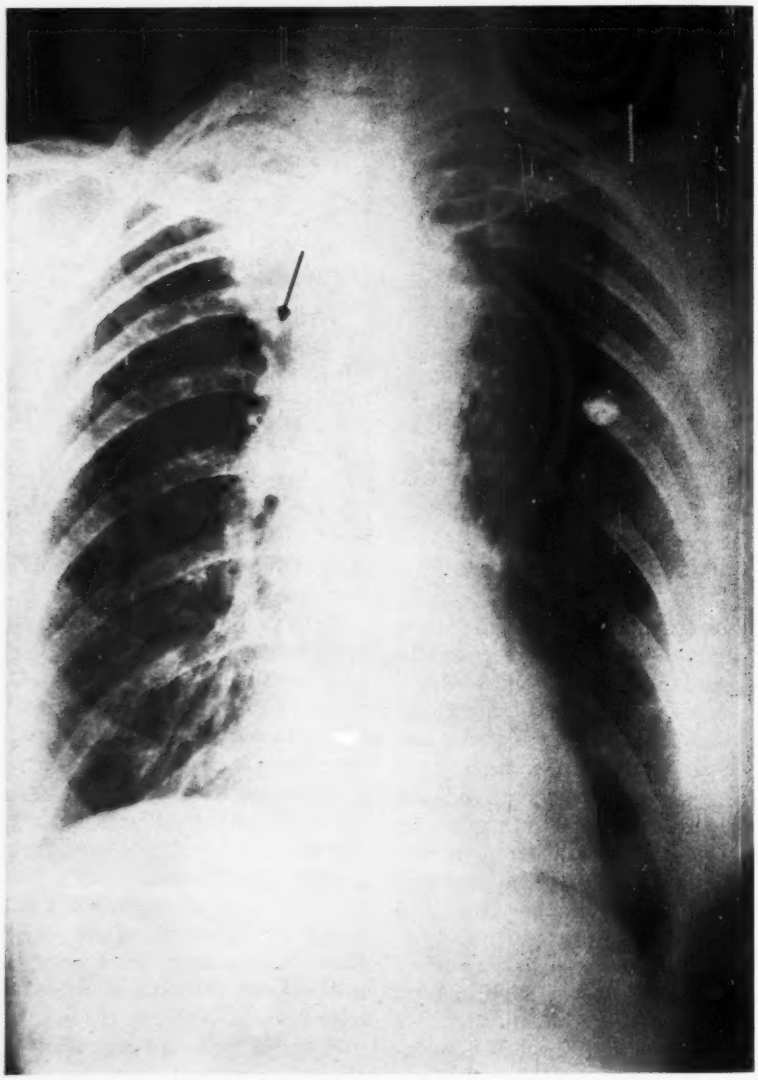

Fig. 5. Case 2. Postero-anterior projection visualizing a wedge-shaped pneumonitis in the right apex. Note the contour of the calcification at the apex of this consolidation. This film was made during the febrile period.

manifestation. Because of this, the case may too readily be relegated to the category of allergic or cardiac asthma. Roentgenologic recognition of the concretion, perhaps with its peripheral wedge-shaped density of atelectasis or infiltration, would forestall idle waste of time and effort to establish a specific protein or remedy a suspected myocarditis. The deception of diagnosis because of a marked bronchorrhea, or because of superimposed infection has already been mentioned. Stone asthma should be considered, then, in cases which might be classified as bronchitis, bronchiectasis, pneumonia, abcess formations,

or infarction. On the basis of broncholiths, infectious processes are seldom widespread, and febrile reactions might, therefore, be

Fig. 6. Photograph of stone expectorated by the patient. Compare its contour with that of the calcification visualized in the film above (Fig. 5).

expected to be less severe than when the bacterial invasion has taken place by virtue of its own virulence. When hemoptysis supervenes, one is prone to brand the patient as actively tuberculous, even though repeated sputum analyses be negative and though other clinical evidence may be lacking. Broncholiths may account for extreme symptomatology in the active or quiescent tuberculous patient, and again the proper recognition of the process is important to establish prompt alleviation of the symptoms. If one were overly tumor-conscious at this stage, there might be an inclination to consider bleeding papillomas or even carcinoma of the tracheo-bronchial tract.

The following two cases are reported. The first exemplifies the most common type of broncholith formation, and the second, probably, the next most frequent basis.

Case 1. (Figs. 1, 2, 3, and 4.) W. H. N., a 54-year-old male, requested medical attention because of hoarseness and a "scratching" sensation in his throat. He stated that periodically, during the past six years, he had been bothered with attacks of a wheezing cough, at times accompanied with slight dyspnea, and associated with a slight amount of mucus and occasionally with blood. On four occasions he had raised definite stones; after the expulsion of each of these he expectorated a "moderate amount" of bright red blood. Otherwise, his sputum was occasionally blood-tinged and he frequently noted a grittiness as he expectorated. He felt very well between

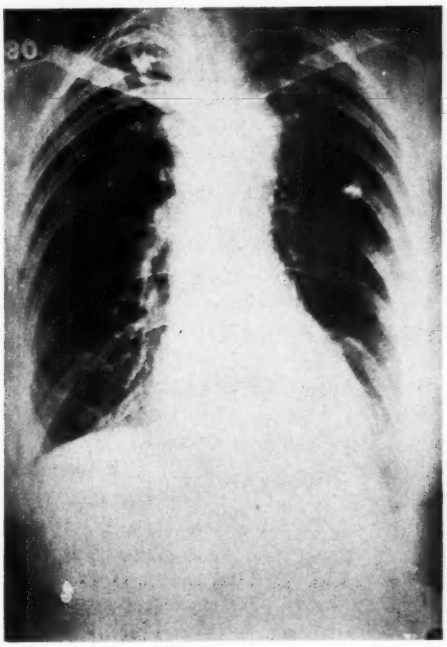

Fig. 7. Postero-anterior projection made some weeks after the expectoration of the stone. Note the clearing of the pneumonitis and the absence of the calcification previously visualized at its apex.

these attacks except for residual hoarseness, which seemed to develop after raising the stones. Some of the coughing spells persisted as long as three months.

The family history was virtually irrelevant; there was no record of tuberculosis nor of other lung conditions among the immediate or distant relatives. Remote past history was likewise of no assistance; the patient stated that he had been in very good health previous to the past six years, except for the usual diseases of childhood.

A physical examination revealed a healthy appearing individual of normal temperature, pulse, and respiration. The general examination was essentially negative except for the larynx, which appeared slightly edematous and pale and showed a content of mucoid material. Fluoroscopic examination of the thorax showed good motion of both domes of the diaphragm. The mediastinal structures did not change their position during either phase of respiration,

and the swallowing function appeared normal. Roentgenography of the chest is represented herewith. The sputum was repeatedly negative for acid-fast organisms but showed many polymorphonuclears and epithelial cells.

Bronchoscopic examination by Dr. Gabriel Tucker showed "a slight thickening of the mucous membrane throughout the tracheo-bronchial tree. Very little abnormal secretion was present. At a point on the external and lateral wall of the right bronchus there was a break in the mucous membrane which was possibly the point through which the broncholith, that the patient coughed up, had been excreted."

Case 2. (Figs. 5, 6, and 7.) This patient was a woman of 60, who had had a tonsillectomy about nine weeks previous to the date of the first chest roentgenography (Fig. 5). Her surgeon had noticed, during the course of the operation, the release of two tonsilliths and their falling into the pharynx. He was able to recover one of these but the other one disappeared. About two weeks after the operation this patient developed a "pneumonia" in the upper lobe on the right side. She maintained a febrile course, with drenching night sweats and a drastic cough, for more than three weeks. Because of the persistence of these symptoms, the roentgenologic study was made. A few days after the first roentgenography, she developed a very severe coughing spell which was climaxed by the expectoration of a lith, a photograph of which is shown in Figure 6. Her symptoms then promptly subsided. Subsequent roentgenography revealed a clearing of the wedge-shaped pneumonitis and the absence of a calcific density which had previously been visualized at the apex of that zone. The size and contour of the lith coincided with that of the calcific density.

SUMMARY

The subject of pneumoliths and broncholiths has been briefly reviewed. Emphasis has been placed upon the statement that the roentgenologist and clinician should consider the possibility of bronchial liths when confronted with a case manifesting profuse bronchial secretions, bronchial spasm, intermittent hemoptysis, or, perhaps, evidence of localized pneumonitis. Differential considerations include, in particular: allergic asthma; cardiac asthma and tuberculosis, but also, chronic bronchitis; bronchiectasis; lung abscesses and pneumonia, and to a lesser degree, benign and malignant neoplasms of the tract.

We are indebted to Dr. Gabriel Tucker for the clinical record in the first case reported, and to Dr. J. H. Vastine for the loan of the second case reported.

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LEONTIASIS OSSEA: A CLINICAL AND ROENTGENOLOGICAL ENTITY

REPORT OF A CASE

By J. H. GEMMELL, M.D., *Philipsburg, Pa.*

HISTORY

THE patient, M. C., a male child of English extraction, aged seven and one-half years, was first referred to the orthopedic clinic of this hospital in June, 1931, with the complaint of a misshapen, enlarged head, curvature of the spine, and general debility. Roentgenograms of the skull and of the bones of the legs were taken, and at this time the family was notified that nothing from an orthopedic standpoint could be done for the child.

In October, 1934, when he was almost eleven years old, the child returned to the out-patient department for more complete study.

The boy was the fourth of seven living children of the mother, now 31 years of age. In addition to the seven children, she had had three other pregnancies, two resulting in miscarriages in early months, and the other resulting in the death of the child at birth, due to injury. All of the other living children are entirely free of physical defect. There is no antecedent history of any defect resembling that of this particular child. The mother had a normal labor in giving birth to the patient, and states that she believed his head to be a little large but that otherwise he seemed normal. He was breast-fed until the age of one year, sat up alone at seven or eight months, and began to walk at about one year.

There is a history of an attack of scarlet fever during the second year, with subsequent drainage from the ears for a long time, with a resultant hearing defect. Also, there was a severe attack of measles at four but with apparent complete recovery. At the age of six, the patient developed an infection in the right hand following an injury by a splinter of wood. This required considerable attention from a surgeon before healing finally took place; it is stated that the boy has had practically no use of the right arm and forearm since. The mother says that she was not impressed with anything particularly unusual about the boy's head, except for size, until the seventh year, when it became noticeably more enlarged and of a peculiar shape, and began to hang over to the left side. The boy began school during that year but had to drop out due to failure to progress in his studies, and because of defective vision and his general physical condition. Since that time he has remained at home continually, and although able to be up and around, he is handicapped physically by the enormity of his head and the paresis of the right arm, and mentally by the defects in hearing and sight, and by inability to obtain any proper training. The child behaves well, is useful at home insofar as he is able, and his only complaint is of occasional headache. Gradual further enlargement of the head has been reported in the past three and one-half years, but there is little apparent change in the general appearance or contour of the skull and face since the examination in 1931.

PHYSICAL EXAMINATION

The child is generally underdeveloped and underweight for age eleven. He walks carefully, with a hunched-over posture and with the head tilted to the left; grossly, the striking features of the case are the size of the head, peculiar facies, thickened prominent clavicles, paresis of the right arm, forearm, and hand, and the marked dorsolumbar scoliosis.

The head is symmetrically enlarged, particularly in the longitudinal axis, and is remarkable for its great weight, apparently out of all proportion to size; the impression in lifting the head is that of moving some solid metallic object. The fronto-

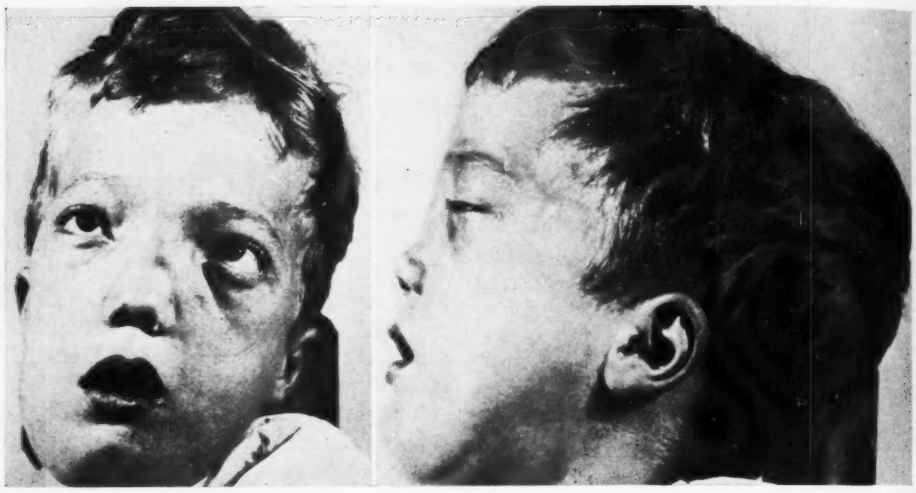

Fig. 1. Fig. 2.

Fig. 1. Note the shape of the head, the peculiar nasal contour, the slight proptosis, and the prominent lower jaw.

Fig. 2. The open mouth is a necessity for respiration.

occipital diameter measures nine inches; the circumference in this plane is twenty-six inches.

The facial features are: eyes set wide apart, both being slightly exophthalmic, a peculiar nasal contour (similar to a saddle nose), the open mouth, and the prominent lower jaw. Inspection of the nose indicates almost complete obliteration of the nasal cavity, so that the boy is forced to keep his mouth open continually for satisfactory respiration. In the mouth, the hard palate is less arched than usual; the tonsils and adenoids are hypertrophied. The ears show no discharge, and there is no tenderness over the mastoids.

General examination of the neck, chest, and abdomen elicits no pathology of the thyroid, heart, or lungs, abdominal contents or genito-urinary system.

Neuro-psychiatric examination by Dr. H. E. Eaton, of Warren, Pennsylvania.—At the present time, the patient presents the picture of an emaciated young boy, barely able to sustain the weight of an enormous head. The right arm is atrophied, the patient being able to move it only slightly. The clavicles are thickened and prominent. There is a marked right dorsal left lumbar scoliosis, with considerable reduction in size of the right chest. Examination of the cranial nerves reveals a marked bilateral nystagmus, though ocular movements are fairly well carried out and the pupils react to light and accommodation. The facial muscles are greatly stretched, though they all appear to be normally activated. He is partially deaf, and bone conduction is much better than air conduction. There is a marked fibrillary tremor of the platysma on the right, possibly due to the exertion required in keeping the head erect. There is considerable atrophy of the right arm and the muscles of the shoulder girdle on the right, and the left shoulder is easily dislocated but returns quickly to its socket with slight manipulation. There is marked diminution of muscle power in the right arm, forearm, and hand, the patient using his left arm to place his right arm and hand where he wishes them. Tendon reflexes are present on both sides but are more active on the left. In addition to the above findings, there is also a very marked myopia.

Psychometric examination gives this boy a mental age of five years, ten months, with an I.Q. of fifty-three. However, this examination does not entirely reveal the

Fig. 3.

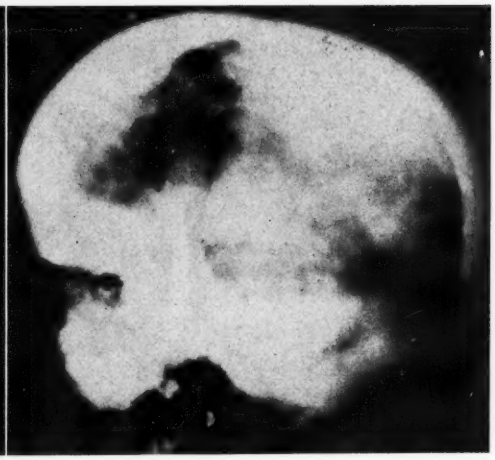

Fig. 4.

Fig. 3. Roentgenogram taken in June, 1931. All the bones of the skull and face are involved in a sclerosing process, obliterating the nasal sinuses and the mastoids.

Fig. 4. Roentgenogram taken in October, 1934. Increased thickness of bones is particularly noticeable in the frontal region and base of the skull. This increase is mostly at the expense of cranial capacity.

child's mental capacity because of his deafness and difficulty in seeing well. He has had little schooling because of his infirmity. However, he appears fairly alert, and talked freely with the examiner, though he was at first fearful that he should be hurt, but, when reassured on this point, cooperated to the best of his ability with all examinations. He appears to recognize his physical difficulty and asked the examiner if he could so something to help him.

In the examiner's opinion, this boy's neurologic findings are probably secondary to the involvement of the bone. The nystagmus can be explained on the basis of involvement of the labyrinth, although better bone conduction than air conduction indicates middle ear involvement, possibly by the same disease. The involvement of the shoulder girdle and arm muscles on the right side is possibly due to injury to the brachial plexus or to nerve roots, and decreased tactile sensibility on the right appears to bear this out.

LABORATORY EXAMINATION

Blood cytology showed red blood cells, 4,200,000; white blood cells, 6,900 per cubic mm., with a hemoglobin of 80 per cent, and with a differential count of neutrophiles, 45 per cent, lymphocytes, 50 per cent, and eosinophiles, 5 per cent.

Blood chemistry showed: sugar, 113 mg.; non-protein nitrogen, 36 mg.; phosphorus, 5.2 mg.; calcium, 10 mg. per 100 c.c. whole blood.

The blood Wassermann was negative.

Urine and spinal fluid examinations were negative. Basal metabolism, 1 per cent. A biopsy was not obtainable.

ROENTGEN EXAMINATION

In 1931, when only the head was examined, all the bones of the skull and face showed excessive symmetrical overgrowth, resulting not only in general enlargement out of proportion to age, but also in extreme thickening. This overgrowth was of a very dense consistency throughout, there being no cystic areas present anywhere. The mandible was thickened and sclerotic. The paranasal sinuses were completely obliterated, as were the mastoids, and the petrous portions of the temporal bones and the entire base of the skull were greatly sclerosed. The sella turcica could scarcely be discerned, apparently due to the piling up of bone in the basal structures and side

Fig. 5.

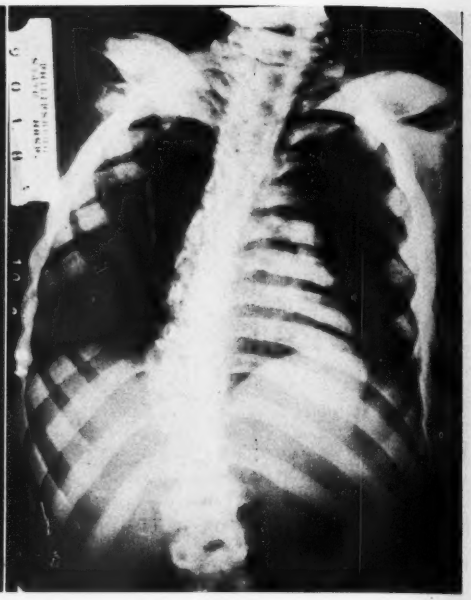

Fig. 6.

Fig. 5. Roentgenogram taken in October, 1934; anteroposterior occipital projection. The disease process appears symmetrical.

Fig. 6. The clavicles are thickened and sclerosed in a fusiform manner, and the ribs are so widened that a diagnosis of lung pathology would be impossible.

walls of the skull in that plane; the sella appeared somewhat shallow and measured 14 millimeters, inside greatest diameter, on the original roentgenogram (Fig. 3).

The examination in October, 1934, was more complete. Contrary to the impression of the mother, the over-all dimensions of the skull showed no increase. However, the progression of the bony overgrowth inwardly was notable. By way of demonstrating this, measurement of the thickness of the frontal bone on the 1934 film (Fig. 4) was 3.5 cm., in comparison to 2.5 cm. on the 1931 film for the same portion of bone, the same focal distance having been used in each case. Thus it would seem that the cranial capacity is diminishing. All the other bones of the skull and face showed proportionate increases in thickness. The sella was less easily seen, and measured only 10 mm. inside diameter. The technic required to obtain the original roentgenogram, reproduced as Figure 4, was 6 seconds, 15 ma., 80 kv.p., 30-inch focal film distance, Bucky diaphragm, using standard film in a casette, with parspeed intensifying screens.

Other portions of the skeleton were involved in this sclerotic process, particularly the ribs and clavicles. The latter were enlarged in a fusiform manner, while the former were more diffusely thickened to such an extent that the rib interspaces were greatly narrowed. There was apparent a beginning sclerosis of the cervical vertebræ, but the dorsal and lumbar regions of the spine were of normal density. Of particular interest was the lack of significant change in the scapula, pelvis, or bones of the extremities, other than the decalcification from disuse of the right upper extremity bones.

There were no cystic areas in any portion of the skeleton, and it is of note that in the skull there was not the "curled, kinky-hair" appearance characteristic of Paget's disease.

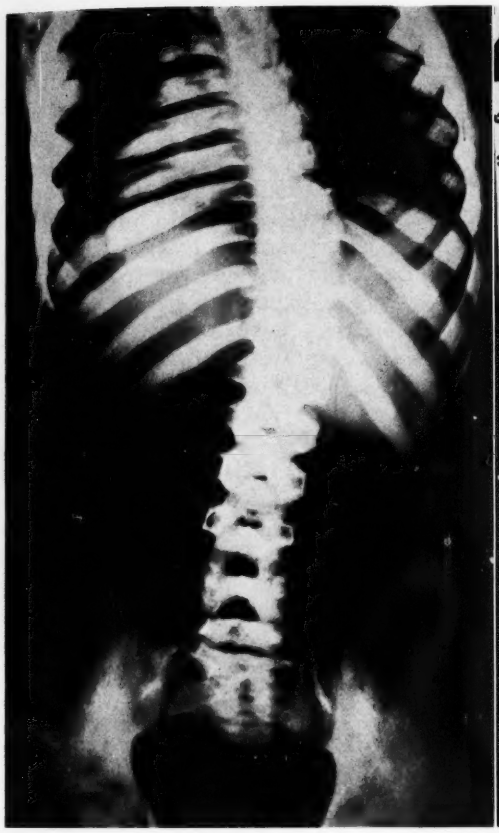

Fig. 7.

Fig. 8.

Fig. 7. Showing the dorso-lumbar curvature. The ribs are almost as dense as the vertebræ. The pelvis is normal.

Fig. 8. The bones of the thighs and legs are normal.

DISCUSSION

Virchow (9) first used the term "leontiasis ossea" in describing the facial and cranial deformities due to hypertrophy of the bones of the face and skull. Although acromegaly and other benign and malignant lesions resulting in facial disfigurement have since been discussed as leontiasis ossea, this terminology has been lately confined to cases in which hyperostosis is the predominant lesion. Knaggs (5) classified a large number of these cases into a creeping periostic variety and a diffuse osteitic variety. The histology of the disease is usually that of osteitis fibrosa, *i.e.*, disappearance of normal bone and replacement at first with fibrous tissue which later ossifies.

In addition to what might be termed pure forms of the disease, are cases in which manifestations of Paget's osteitis deformans or occasionally perhaps von Recklinghausen's osteitis fibrosa cystica are present, as well as the hyperostosis of the cranial and facial bones ascribed to leontiasis. Indeed, Freedman (3) suggests that leontiasis ossea can be considered a localized form of either of these diseases since the histologic findings in all are similar.

However, it must not be overlooked that typical cases of Paget's disease or von Recklinghausen's disease do not possess the

profound hyperostoses of the skull and particularly of the facial bones characteristic of leontiasis. Harvey Smith (8) describes typical Paget's disease of the skull as "undue prominence of the frontal bones, and non-involvement of the face. The bone is several times normal in thickness, without encroaching upon the skull cavity. There is a finely porous outer table covered with small nodules of bone." The classical picture of von Recklinghausen's disease, when it involves the skull, is that of general bone decalcification, many cystic areas, and an irregular mottled sclerosing process. The facial bones are not involved. It might be mentioned too that these diseases differ from each other considerably not only from roentgenologic and clinical standpoints, but also from the etiologic standpoint, in that osteitis fibrosa cystica is a disease associated with parathyroid dysfunction, and is frequently characterized by typical blood calcium and phosphorus changes, while Paget's disease is not thus established. In a recent editorial, Doub (2) discussed the etiology of von Recklinghausen's disease. And, on the other hand, it may be repeated that so-called pure forms of leontiasis ossea may be manifested entirely as proliferative sclerosing bony processes involving principally the face and skull, and show no distinctive resemblance roentgenologically or clinically to these other diseases.

Following are mentioned a few case reports recorded in recent literature which illustrate some of the points under discussion. Hamburger and Nachlas (4) describe a case of a woman 56 years of age, with hyperostotic deposits obliterating the maxillary sinuses and involving the facial bones generally, and also with apparent Paget's disease of the cranial vault and of the tibiæ. Ivemy's (6) unusual case is that of a boy eleven years of age, with an asymmetrical leontiasis affecting mainly the right side of the face, and with other profound changes in the skull and throughout the skeleton; in some areas, the bony change is reported as resembling Paget's disease, and in others, as von Recklinghausen's disease. Freedman (3) also cites a case of asymmetrical hyperostosis of the right side of the face with a resemblance to Paget's disease in the other portion of the skull. Capon (1) describes his case as the diffuse osteitic form, the hyperostosis affecting the upper maxilla, the frontal bone, and the anterior portion of the base of the skull, chiefly on the left side. Hyperostosis is the predominant feature in this case. The impression gathered upon reviewing these cases is that we are dealing with a definite clinical and roentgenologic picture, leontiasis ossea, to which there may or may not be added evidence of other bone dyscrasia—usually a typical Paget's disease. And it would also seem that the signs of leontiasis appeared first.

Reference need not be made to typical cases of Paget's disease. LeWald (7), in discussing Freedman's paper, mentioned a study of 69 cases in which not a single one showed the involvement of the facial bones peculiar to leontiasis.

In the present case, and in the cases just referred to in which blood serum calcium determinations were made, the reported lack of significant increase of this constituent in the blood indicates in a general way a differentiation from the majority of cases of osteitis fibrosa cystica.

The etiology of leontiasis ossea is not definitely known, but trauma, syphilis, nonspecific infection, and endocrine pathology each has been suggested as a causative factor. It is impossible to determine in this case whether or not the disease was present at birth; it might possibly have originated as a result of the persistent otitis media which existed during the second year. There is no history of trauma to the head, and evidence of syphilis and endocrine disturbance is lacking.

SUMMARY

Granted, that there is a pathologic resemblance of leontiasis ossea to Paget's osteitis deformans and to von Recklinghausen's osteitis fibrosa cystica, yet

(1) Cases of leontiasis occur having none of the usual clinical or roentgenologic fea-

tures of either Paget's or von Recklinghausen's disease.

(2) There are cases of leontiasis having associated bone lesions suggestive of these other diseases in atypical forms.

(3) Innumerable cases of Paget's disease exist without any evidence of leontiasis.

(4) Many cases of von Recklinghausen's disease occur without leontiasis; they are related to parathyroid dysfunction, and usually show variation from the normal values for calcium and phosphorus in the blood.

(5) There is apparently something fundamentally different and distinct in cases having this diffuse hyperostosis of the skull and facial bones, regardless of the added presence or absence of bone lesions suggesting other disease.

CONCLUSION

It is contended that leontiasis ossea may still be classified as a clinical and roentgenologic entity.

The present case is offered as a pure form of the disease.

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THE RÔLE OF THE ROENTGENOLOGIST IN THE PROPER MANAGEMENT OF PLEURAL ADHESIONS PREVENTING EFFECTIVE PNEUMOTHORAX COLLAPSE

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THE continuance of an ineffective artificial pneumothorax attempted over a long period of time is useless. The dangers of extension of the tuberculous process from the open cavity into the affected lung, as well as cross-firing into the so-called good lung, are great. Hemorrhages cannot be prevented, and spontaneous pneumothorax and subsequent empyema are especially menacing in cavities localized just below the surface of the lung which are held open by adhesions. The high-pressure pneumothorax, with and without artificial fixation of the mediastinum, is sometimes successful in such cases if administered with small doses of air given at short intervals, but its possible dangers cannot be underestimated. A tear near the thoracic wall, followed by uncontrollable hemorrhages into the pleural sac, or a tear into the lung tissue, followed by spontaneous pneumothorax, cannot be prevented, even if the best technical skill is assured.

Therefore, electrosurgical severance of pleural adhesions should be much more often practised than heretofore, in order to convert an ineffective artificial pneumothorax into an effective one. The open method by thoracotomy is dangerous. The percentage of pleural empyemas in such cases is high, and the shock to the weakened tuberculous individual is, in many instances, too severe to justify such a procedure. Since thoracoscopy is relatively harmless, intrapleural closed pneumolysis should be the treatment of choice in such instances.

WHAT ARE THE ROENTGENOLOGIC INDICATIONS FOR THIS TYPE OF OPERATION? OF WHAT AID COULD THE ROENTGENOLOGIST BE TO THE THORACOSCOPIST?

Every experienced phthisiologist knows that pleural adhesions are present in almost every case of artificial pneumothorax. They are almost physiologic, and disappear or give way in many instances even under negative intrapleural pressure. But the picture changes as soon as the adhesions become organized, forming fibrous strings, cords, bands, or fans, which prevent the tuberculous cavity from collapsing, or which, by contraction, cause a re-expansion of the collapsed lung and reopening of the cavity already closed by an effective collapse. Here, the aid of the roentgenologist is invaluable. It is up to him to study carefully large series of chest films taken at short intervals, and to describe every single adhesion and its character, often resorting to lateral films taken at different angles, in order to differentiate between cords and fans. These latter frequently prove to be unsuitable for closed intrapleural pneumolysis on account of the danger of lung tissue or cavities extending into the adhesions. He will often be able to call the phthisiologist's attention to the fact that, in his attempt to stretch pleural adhesions, he had accomplished nothing but a mediastinal hernia with its consequences, such as disturbance in the lesser circulation, downward pressure of the diaphragm, etc. He will try to determine fluoroscopically, if possible, the character and amount of fluid in the pleural sac, and advise aspiration if it interferes, in his opinion, with the proper visualization of the adhesions in case of pneumolysis. He will study the contralateral lung with greater care, and advise for or against pneumolysis, depending upon the extent and severity of the contralateral tuberculous process. And, finally, he will be careful in committing himself as to the character and number of pleural adhesions, leaving the decision in questionable cases rather to the thoracoscopist. Thoracoscopically,

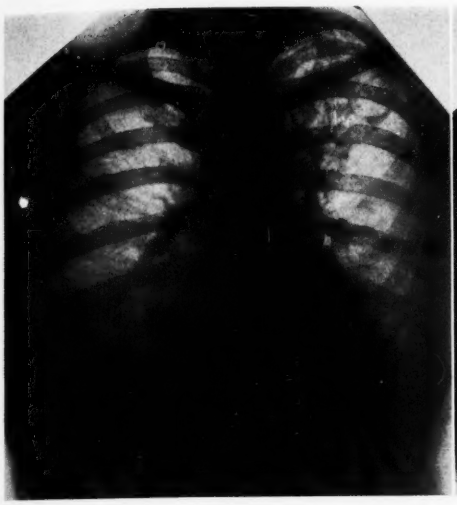

Fig. 1. Case 1. Roentgenogram showing infraclavicular cavity on left.

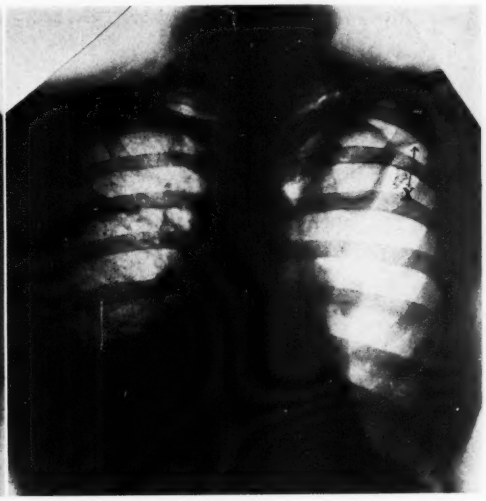

Fig. 2. Case 1. Ineffective pneumothorax; cavity larger than before.

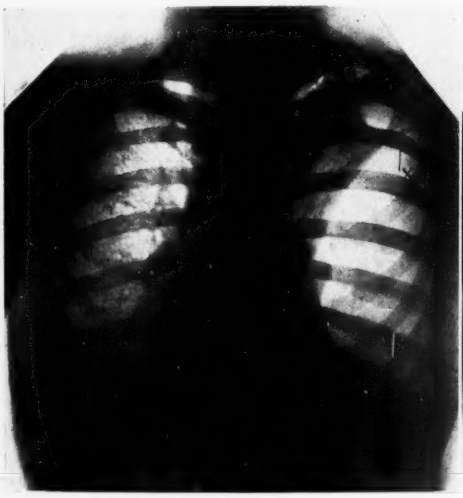

Fig. 3. Case 1. Two weeks after partial pneumolysis. Lateral adhesion (*X*) was not severed; cavity is diminished in size.

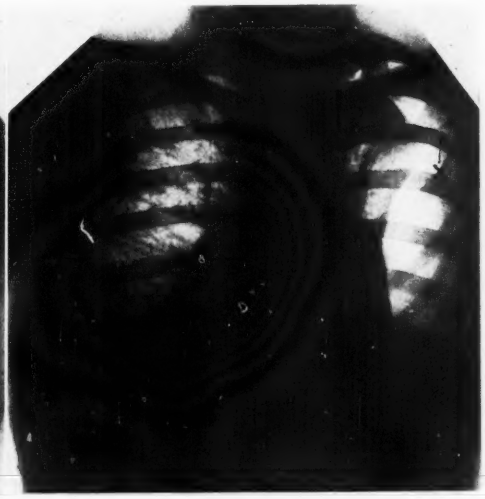

Fig. 4. Case 1. Cavity closed in spite of lateral adhesion (*X*) not having been severed.

it is amazing how often one finds that there are many more adhesions, often of quite a different character, than were observed roentgenologically. For instance, as Matson has pointed out, the floor of an uncollapsed cavity looks sometimes exactly like a pleural adhesion. The thoracoscopist, relying upon such an erroneous diagnosis, would cause an irreparable damage to the patient.

It will often be impossible to answer the important question, "Which of several adhesions is chiefly responsible for the failure of the cavity to collapse?" It is a well-known fact that the closure of such cavities, after only partial pneumolysis,

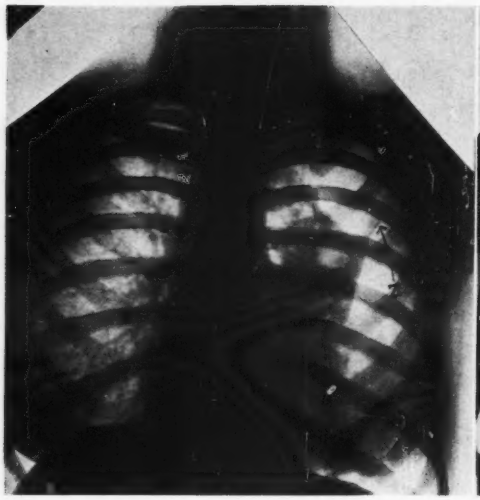

Fig. 5. Case 2. Ineffective pnemothorax, apical and lateral adhesions.

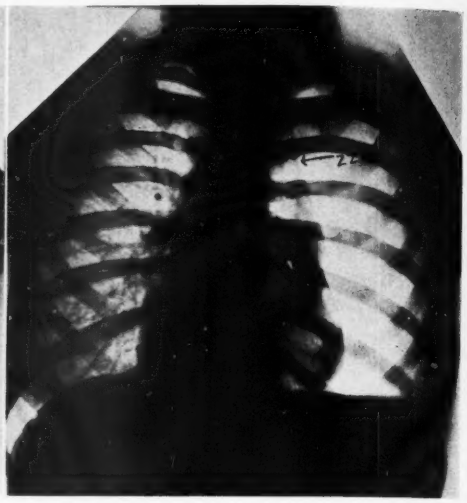

Fig. 6. Case 2. Lateral adhesion severed, apical adhesion untouched, and cavity closed.

is not rare if the adhesions are apical, string- or cord-like, and left intact, because they are easily stretched. Also, in exceptional cases, the thoracoscopist can accomplish a favorable result if he omits severance of adhesions which, judging from the x-ray appearance, he should have cauterized in order to guarantee the obliteration of the cavity. The following is an example.

A widow, 35 years of age, mother of two children, with a positive sputum and a large infraclavicular cavity on the left (Fig. 1), was given high-pressure pneumothorax without success (Fig. 2). The cavity, after pneumothorax treatment, seemed to be even larger than before. The patient was sent to me from a sanatorium, with the request that I sever the pleural adhesions electrosurgically. I was asked to pay special attention to one thick lateral adhesion (*X*). This, from the x-ray appearance, seemed to be mainly responsible for the failure of the cavity to collapse. Thoracoscopically, I found eight adhesions, seven apical ones posteriorly, and one broad lateral band (*X*). It was relatively easy, by the two-puncture method, to sever the seven apical strings and cords, but the lateral broad band (*X*) showed, on close transillumination, a definite pulsation which was not transmitted from the heart but was due to a distinctly visible vessel within. I decided, therefore, because of the danger of an intrapleural hemorrhage, to leave it alone. The patient was discharged from the hospital after a week, and the physician at the sanatorium was advised to continue artificial pneumothorax with small doses of air to be given at short intervals. The next roentgenogram (Fig. 3), taken two weeks later, showed, much to my surprise, the cavity greatly diminished in size, and the film taken four weeks after pneumolysis revealed a complete closure of the cavity (Fig. 4). The sputum became negative immediately and has remained so since. The patient is now doing housework and receives ambulant pneumothorax treatment from her family physician.

The simple relaxation of the cavity, by cauterization of the minor apical adhesions alone, was sufficient to obtain an effective pneumothorax in spite of a broad lateral band left untouched.

In contrast to this case I call attention to two films of another patient (Figs. 5 and 6), one taken before and one after closed intrapleural pneumolysis. In this case the

severance of the lateral band (*Z*) alone was sufficient to secure an ideal collapse of the cavity without touching the apical adhesion (*ZZ*).

SUMMARY

The roentgenologist's aid is of great value in cases of incomplete collapse of tuberculous cavities held open by pleural adhesions. In questionable cases, the roentgenologist should not commit himself, because the roentgenogram differs frequently from the thoracoscopic picture.

A case is presented in which a lateral broad adhesion of a vascular character, which, from the roentgen standpoint, appeared to be alone responsible for the failure to close a cavity, was left untouched without being severed. After cauterization of minor, apparently unimportant apical adhesions, this band stretched sufficiently to permit a complete closure of the cavity. Films of a contrast case are presented.

The final decision as to the procedure of intrapleural closed surgery should not be made on roentgenologic evidence alone, but also by thoracoscopy.

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A HISTOLOGIC STUDY OF THE EFFECTS OF X-RAYS ON FROG SKIN

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IN 1903, Allen (1) recorded that, following x-radiation, the skin of a man showed an increase of connective tissue in the corium. The epidermis had disappeared in some places but in other sections it sent processes downward into the corium. Necrosis of tissue was observed under the epidermis. In another of his experiments, the skin of a frog was irradiated for 57 minutes and immediately fixed in Zenker's solution. An increased vascularity was noted. The following year Una (8) reported that irradiation caused a thickened epidermis which gradually thinned. Collagenous material increased in thickness and the intimæ of capillaries were closed by thickened walls. Telangiectases were observed. The squamous cells sloughed off and all epidermal appendages atrophied. This article gives a good review of the meager literature until 1906.

Porter and White (7) reported that x-radiation produced growths of finger-like projections into the corium, along with dilated vessels, swollen cells, and a disappearing epidermis. The corium contained dilated subpapillary vessels filled with blood. The walls of the enlarged vessels were thicker. In some x-rayed specimens, it was difficult to determine where the corium stopped and the epidermis began. Lack of pigmentation was associated with early x-ray burns. It was hypothesized that the first reaction was on the vascular supply, which caused a thrombosis of the vessels. This was followed by an ulceration, and after four days it was observed that leukocytes had filled the vessels, but, after thirty days had elapsed, the walls of the old vessels had thickened and new capillaries were observed.

Wolbach (9) found that dilated capillaries and telangiectases in a rarified corium were quite common results of irradiation of human skin. Various stages of dilation were observed in the capillaries, and some were even obliterated by proliferation of the endothelium. Elastic and collagenous fibers were increased, the walls of the vessels were thickened, and although the epidermis seemed to be thickened the horny layer was lacking. The downgrowths were very pronounced and the tissue immediately below the epidermis became loose-textured, showing imperfect repair and lesions of the vascular system. The deeper connective tissue became much denser and many large cells were noticeable. Wolbach found that tumors appeared on human subjects several years after irradiation and after the establishment of severe chronic changes of the skin characterized by great thickening of the tissue, hyperkeratosis, and the loss of the epidermal appendages. Downward growths from the epidermis preserved its continuity by obtaining nourishment from deeper connective tissue. The obliteration of the capillaries was probably due to the swelling of the corium. Ulcerations were present only in places where the degenerated area was too large to be bridged by the epidermis. The downgrowths show the typical properties and cells of the epidermoid carcinoma. It is thought that the carcinoma cell is one isolated from the rest of the body through gradual withdrawal of nutrition owing to successive obliteration of vessels, and that the cells thus gradually acquired a greater capacity for securing nutrition and finally became capable of living at the expense of other cells. These experiments confirmed the results gained by previous workers whose articles are briefly summarized in this article.

Del Buono (2) concluded that the first reaction of irradiation was on the vascular system, causing dilation of the minute ves-

Fig. 1. Normal frog skin (× 250).

Fig. 2. Five days after three erythema doses (× 250).

Fig. 3. Twelve days after three erythema doses (× 750).

Fig. 4. Twenty-two days after three erythema doses (× 250).

sels soon after exposure. The younger cells were injured so that their repair took a long time. The result depended on the state or condition of the patient, the young being more sensitive. Latent burns were sometimes noticed although earlier signs were absent. Ewing (3) also observed that growing cells were the most sensitive, while the squamous cells were least so. Swelling of the tissue cells was probably due to the nuclear cytoplasm imbibing water. Mitosis was reduced with abnormal forms appearing. Lymphocytes and plasma cells were increased in the irradiated areas. Heavy irradiation caused mucinous degeneration of collagen fibrils, fragmentation of muscle fibers, hemorrhage of capillaries, and then the actual growth of fibroblasts with the development of new capillaries. The membranes around fat particles were ruptured, allowing the contents to escape.

Conflicting evidence was added when there was found to be no increase in the elastic fibers of mouse skin following irradiation. Also, irradiated carrots proved toxic to guinea pigs, and a theory was propounded that the results obtained in the skin were due to an avitaminosis and not to a direct attack on the cells.

During 1929, Pohle (5) produced histologic evidence that irradiation caused reserve capillaries to open. These vessels remained enlarged as their walls thickened. Later, Jacobson and Waddell (4) x-rayed rats and studied the skin tissue. The horny layer was observed to come off from the epidermis, and the latter to become loosened from the corium. The collagenous fibers of the corium became swollen and fused. Leukocytes digested the area killed, leaving an open vacuole. Intercellular bridges disappeared. Pohle and Bunting (6) have confirmed these results. Excellent plates which show the effects of irradiation on the skin are presented in the last two experiments mentioned.

EXPERIMENTAL PROCEDURE

The following report gives an account of the action of the rays on frog skin and a theory accounting for this action. Frogs were used because the average sections of skin are much more uniform than those from human subjects, and because these animals are easier to control.

Three female frogs were tied on their backs and their white undersides irradiated with an unfiltered beam from an x-ray tube (W target) with a potential of 100 kv. and a current of 5 milliamperes. The focal distance was 8 inches. The machine had double disc mechanical rectification. Frog *A* was kept as a control; *B* was irradiated for 3 minutes, 36 seconds, comparable to three human erythema doses; *C* for 6 minutes or five erythema doses, and *D* for 7 minutes, 24 seconds, or seven erythema doses. Millimeter-square sections of skin were cut from the irradiated parts at various time intervals and fixed in Zenker's solution. Histologic slides were made and stained with hematoxylin-eosin dyes.

The normal skin consists of the epidermis, which has squamous, cuboidal, and columnar cells, and the corium, which first shows the stratum spongiosum, a loose layer containing glands, blood vessels, pigment cells, and then the stratum compactum, a dense layer of connective tissue (Fig. 1).

Fused muscular fibers were noticed in the skin of frog *B* five days after irradiation. The corium was loosened from the epidermis and became quite wavy. It appeared that the proteins had become coagulated (Fig. 2). Twelve days after irradiation the skin of the same frog showed marked degeneration in the stratum spongiosum, and the vessel walls became enlarged. The columnar cells were becoming less prominent and the outer cells of the epidermis were dying (Fig. 3).

Regeneration of the skin tissue of *B* became rather noticeable 22 days after irradiation, for the epidermis appeared to be thickening and sending processes into the corium. However, the stratum spongiosum was definitely degenerated and the outer cells of the epidermis seemed to be swelling (Fig. 4). Degeneration of the epidermis could not be doubted after 61 days. It was much thinner and no columnar cells

Fig. 5. Sixty-one days after three erythema doses (× 250).

Fig. 6. Twelve days after five erythema doses (× 400).

Fig. 7. Sixty-eight days after five erythema doses (× 200).

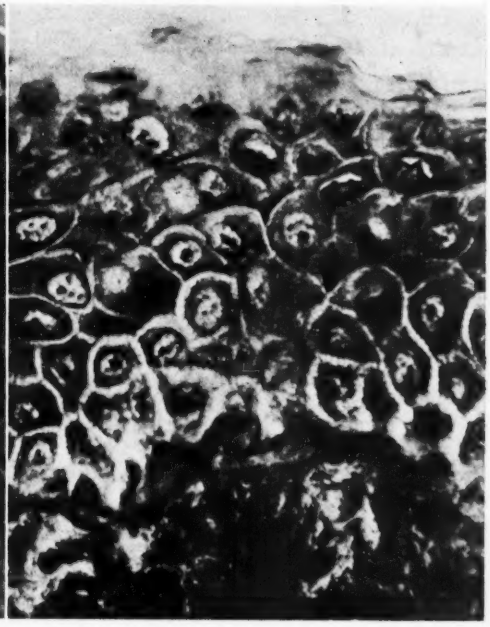

Fig. 8. Twenty-two days after seven erythema doses (× 750).

could be found, and, contrary to previous observations, the intercellular bridges were more defined between the larger cells that remained. Vascularization became more pronounced between the corium and the epidermis. The lower part of the corium was much heavier and many new cells could be found about the minute vessels. The walls of the vessels were definitely thicker and the vascular system was increased. Pigmentation was also more pronounced (Fig. 5).

The skin of frog *C*, which had received five erythema doses twelve days before, gave definite signs that the corium was tearing away from the epidermis, leaving degenerated cells between. The squamous cells were sloughing off and the columnar cells were becoming granular in nature (Fig. 6). After sixty-eight days, all of the columnar cells had disappeared and the epidermis had decreased in thickness. The upper layer of the corium exhibited an intensely proliferated area of growth, having many cell nuclei as previously described. The entire corium had thickened as before and the increased vascularization was also present (Fig. 7). Under high magnification the epidermal cuboidal cells appeared to be fused together, although those that were more normal had very pronounced intercellular bridges. Some mitotic figures were visible.

Frog *D* died twenty-two days after seven erythema doses had been applied. At this time all columnar cells had disappeared and the intercellular bridges were intensified. There seemed to be no regeneration at this time as there was in the case of *B*. Vascularity had increased and the corium had thickened. The granulations in the cell nucleus appeared to be moving to the outer portions of the nuclei (Fig. 8). These observations in general confirm the results of previous investigators, and demonstrate that different dosages will give different effects. The hardiness of the individual cell enters into the reaction, as has been shown by x-radiation of different bacteria, and by determining their death rate under different dosages.

CONCLUSION

The present investigator believes that the effects described are due directly to the action of the x-rays on the cells or to the action of the secondary cathode rays produced by the primary beam impinging on the tissue. According to the most modern viewpoint, "H" substance (10) is liberated quite slowly from the injured cells during a long period of time, causing the capillaries to dilate. Because of this gradual seepage of "H" substance, the dilation persists, and the power of contracting to the original state is lost as other cells grow and fill the stretched network forming the walls of the vessels. This action undoubtedly thickens the walls and makes them less permeable to the nutritive elements that are necessary for the growth of the tissue cells. Gradually some die while others acquire abnormal forms of growth. X-ray shock and increased nitrogen in the urine soon after irradiation might also be explained by the sudden release of "H" substance in more sensitive tissue.

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THE EFFECTS OF X-RAYS ON THE DEVELOPING CHICK

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INTRODUCTION

DURING the course of the investigation of the effects of roentgen rays on the origin and development of the gonads in the chick, certain developmental anomalies and malformations have appeared in the material studied. It became apparent that such irradiation had affected the vitality and hatchability of the eggs, and that further study was indicated. The results of these observations constitute the material of this paper.

APPARATUS

The x-ray machine[1] used was mechanically rectified and provided with a Landauer roentgenometer. A Universal Cooledge therapy tube was used. The set-up of the machine for the entire experiment was as follows: The kilovolt meter was set at 96, which delivered 112 peak kilovolts as measured by the sphere gap; the milliammeter was set at 6 milliamperes; the focal distance was 10 inches; the filter was equivalent to 4 mm. aluminum; the roentgenometer was kept at 3.2 microamperes, which, by calculation, gave 0.6 r per second. The desired r, or dosage, was obtained by varying the time of exposure.

A specially designed irradiation box was used for this experiment. In it was incorporated a portable incubator, a candler, and a scattered-ray absorber.

An electrically heated incubator was used for this work. It was provided with a reliable thermostat, humidity equipment, and a means of ventilation. The temperature was kept at 103° F., with a variation less than 0.2° F., the humidity being kept at 50 to 60 per cent. The eggs were turned twice in 24 hours.

MATERIAL AND METHODS

Eggs of white and brown Leghorn chickens were used for this experiment. All were incubated for a certain period of time before they were irradiated. The age of the embryo varied from 19 to 243 hours incubation. The vitality of all eggs was established with the aid of the candler before irradiation. Controls were kept for each experiment. With the exception of irradiation, the control eggs were subjected to identically the same treatment as were the experimental eggs.

In the early part of the experiment it seemed necessary to determine the effect of the shell and shell membranes on the x-rays. Out of the total of 572 eggs, 63 were irradiated through a window cut in the shell and its membranes. After irradiation, the opening was sealed and the eggs incubated. The rest of the eggs were x-rayed with their shells intact. Another attempt was made by measuring the rays of known amount with a Victoreen r-meter after they had passed through the shell and the shell membranes. The results will be discussed later in the paper.

RESULTS

The results of this experiment can be divided into the following groups: (1) Embryos that died in the shell at or shortly after irradiation; (2) chicks that died after considerable development, which are hereafter spoken of as 15 ± days old chicks; (3) chicks that died in the shell at the completion of development, or full term; (4) chicks that hatched; (5) chicks that showed various degrees of anomalies and deformities.

EMBRYOS THAT DIED IN THE SHELL AT OR SHORTLY AFTER IRRADIATION

These constitute a large group, 255 out of a total of 572 eggs. All were incubated prior to radiation. The age varied from 19 to 240 hours. The dosage used was from 60 to 600 r. All were known to be viable

[1] *Type C* model, made by the Standard X-ray Company.

before they were placed on the x-ray machine.

CHICKS DYING IN THE SHELL AT 15 ± DAYS

In this group of 68 eggs were included chicks that had undergone development for several days after irradiation. Gross examination of such chicks reveals no cause that would explain their death. There were no signs of malposition, strangulation, or drowning. The majority are, to all appearance, normal chicks. Some show various sorts of deformities which will be described later in this paper. The age of this group varied from 19 to 73 hours. The dosage was from 60 to 400 r.

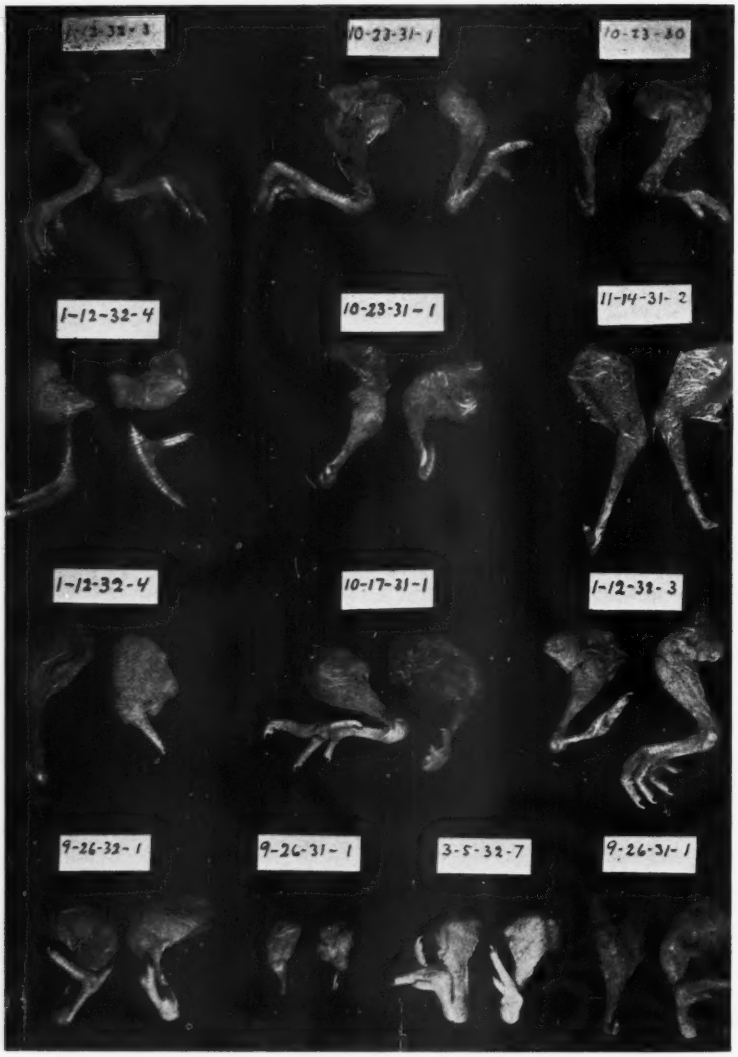

Fig. 1. Deformed lower extremities.

CHICKS DEAD IN THE SHELL AT FULL TERM

Of these there were 138 eggs. The age varied from 19 to 73 hours, and the dosage was 60 to 400 r. The chicks did not have the ability to break the shell and hatch. A few made an opening in the shell but failed

to extricate themselves from it. This condition is not caused by malposition, strangling, or drowning, but it is probably due to the lack of vitality of the chick. In approximately 70 per cent of these specimens, the yolk sac was protruding from the abdominal wall. It may vary from a slight protrusion to a completely extended yolk sac. More than 30 per cent of these chicks are smaller in size than the controls at the time of hatching. Abnormalities of various sorts are common in these chicks.

Fig 2. (*left*) Deformed head of full-term. Fig. 3. (*right*) Head with skin removed; (*a*) normal, (*b*) deformed.

CHICKS THAT HATCHED

There were 111 birds in this group. The age varied from 19 to 240 hours, and the dosage was from 40 to 600 r. They were equivalent in size to the controls. Approximately 83 per cent of the hatched chicks appeared normal in development and behavior; the remaining 17 per cent were deformed. Most of the apparently normal chicks were raised to maturity for further observation.

ABNORMALLY DEVELOPED CHICKS

Chicks having abnormal development may be divided into four subdivisions: (1) Chicks with deformed inferior extremities. (2) Chicks with deformed heads. (3) Paralyzed birds. (4) Chicks with miscellaneous deformities.

Deformed Inferior Extremities.—To this group belonged 34 chicks. They were obtained from three sources: hatched chicks; chicks dead in the shell at full term, and chicks dead in the shell at 15± days of incubation. The abnormalities vary from distorted toes on one or both feet to a total lack of one or both feet (Fig. 1). That part of the extremity above the missing members was, to all appearance, normally developed. The reduction in the number and size of the toes was the most common finding in this group of abnormalities. In many cases only one toe or part of a toe remained attached to the metatarsal, tarsal, or in some cases, directly to the tibia. The average age was 53 hours; 48 hours incubation gave the largest number of teratology. The dosage in all, except one specimen, was 400 r. In the exception, the dosage was 240 r and the age 170 hours.

Chicks with Deformed Heads (*None of These Hatched*).—These deformities appeared to be similar to hydrocephalic malformations, produced by x-rays in mammals (Job, Leibold, and Fitzmaurice, 10). Both the brain and the cranium were enlarged in all dimensions, but particularly in the dorso-ventral axis (Figs. 2 and 3). This combination was found in chicks dead in the shell at full term, and at 15± days of incubation. The size of the head varied from slight enlargement to that shown in Figures 2 and 3. The enlargement was not symmetrical in all cases, in some specimens one half of the brain was normal in size and the other half was considerably enlarged. There were eight symmetrically developed hydrocephalic specimens. The

average age for this deformity was 128 hours, and the average dosage was 217 r.

Paralysis of the Inferior Extremities.—The chicks belonging to this group were unable to walk or stand upright; they moved about by propelling themselves with their wings and beak. Both lower extremities were involved. They were limp, and the toes were usually flexed. The chicks died early owing to their inability to secure sufficient food and water. With some care they could be made to survive, but would show no improvement except in the efficiency of using their wings and beak as a means of locomotion. Other deformities were not usually associated with instances of paralysis, except in one specimen having a distorted neck or torticollis. There were ten paralytic specimens. The average incubation period was 70 hours, and the average dosage was 400 r.

Miscellaneous Deformities.—Under this heading were included abnormalities which were known to occur also in the controls. Of such, altogether six in number, there were cases of polydactylism, acrania, and bill and wing deformities. Polydactylism is the most common deformity of this group, but such instances were not more common in the x-rayed material than in the controls. The acrania cases lack the calvarium; the brain was present but small in size. The bill deformities were of various kinds; usually the lower jaw was affected—it may be shorter than the upper jaw or it may be misplaced. Wing deformities were rare among chicks in this flock. Only one case was recorded in which the metacarpals assumed an abnormal relationship to the radio-ulnar junction, which resulted in turning the tip of the wing outward.

DISCUSSION

The lethal dose of x-ray for white and brown Leghorn chick embryos, with the setup used in these experiments, was close to 600 r. This was apparent by the fact that, out of 29 eggs irradiated with 600 r, only one chick hatched. The age was variable, ranging from 46 to 100 hours.

The effect of the shell and shell membranes on the values of incident x-rays was ascertained in two ways. First, in 63 eggs the shell and its membranes were removed prior to exposure to x-ray. After irradiation the eggs were sealed and allowed to continue incubation. The results obtained from eggs so treated parallel those in which the shell was left intact. The second approach was a direct measurement of rays before and after they had passed through the shell and its membranes by a Victoreen r-meter. By this method it was found that there was a loss of 0.2 r per minute in ordinary dense shells, and 0.4 r per minute in porous shells. This meant that in a dosage of 600 r there was a loss of from 3.33 to 6.66 r. Since very few eggs have porous shells, this loss was very small and not detectable by the first method. Strangeways and Fell (13) report absorption of incident rays by the egg shell to be approximately 40 per cent. This method of measurement or of the calculation was not given in their paper. In passing x-rays through the entire eggs, the Victoreen r-meter showed a loss of 3 r per minute. In view of this, it is difficult to understand the results obtained by the above authors.

TABLE I.—COMPARISON OF X-RAYED (572) AND CONTROL (61) EGGS

Results	Irradiated		Controls		Difference in Per cent
	No.	Per cent	No.	Per cent	
No development	255	45	12	20	25
Hatched	111	20	41	66	46
Death in shell at full term	138	24	5	8	16
Death in shell at 15± days	68	11.9	1	1.6	10.3
Paralysis	10	9	None	None	9
Deformed inferior extremity	34	6	None	None	6
Head deformity	8	1.4	None	None	1.4
Miscellaneous deformities	6	1	3	4.5	−3.5

In Table I, the results of irradiated and control eggs are summarized and the variations expressed in percentages. It will be seen that 45 per cent of all the fertile eggs receiving radiation failed to develop, whereas only 20 per cent of the control eggs failed to develop. The latter 20 per cent is the highest percentage of failure of development of control eggs that have been obtained in this laboratory. This was obtained during the winter period when viability is normally at the lowest.

The best results have reached 100 per cent development. The average obtained in this laboratory as well as that obtained at the supply station, varied between 5–10 per cent. The lowest developmental rate of the control eggs are used here because of the fact that most of this work was done during the winter season.

In the case of embryos dying in the shell at or shortly after radiation, it can be shown that the effect of x-rays on the embryo varies directly with the dosage and inversely with the age (Tables II and III). The same is true of all the groups of the anomalies obtained in this experiment, except that the results are more reliable in cases in which there are a larger amount of experimental animals. In individual cases as well as in several of the groups, considerable variation exists in the correlation between results obtained, dosage, and age of the embryo.

A more or less definite correlation was found only in two groups—deformed inferior extremities and paralyzed chicks. In case of deformed inferior extremities, the age was 48 hours, with a dosage of 400 r, and, in case of paralysis, approximately 70 hours incubation with 400 r, which may be regarded as critical periods in the development of the inferior extremities of the chick.

Heim (9) reports a reduction in size of irradiated chicks two-thirds of the normal. In our chicks, in those which died in the shell at full term, there was a protruding yolk sac in 70 per cent and reduction in size in 30 per cent of cases. In the irradiated chicks that normally were able to hatch, no appreciable reduction in size and weight was noticeable. The above discrepancy in the inclusion of the yolk sac and the lesser size was very likely due to the chicks having died in the shell a day or two prior to hatching time and not to the effect of x-rays.

TABLE II.—AGE 50 HOURS. DOSAGE VARIABLE

Dosage	No. Eggs Rayed	No. Eggs Dead	Percentage of Dead
80	5	1	20
240	27	7	26
400	131	70	53
600	14	14	100

TABLE III.—DOSAGE 400 r. AGE VARIABLE

Age	No. Eggs Rayed	No. Eggs Dead	Percentage of Dead
19	20	19	95
20– 30	26	21	80
31– 48	111	58	52
49– 80	138	54	39
81–140	24	9	39

SUMMARY

Five hundred and seventy-two hens' eggs have been subjected to the effects of x-rays at dosages varying from 40 to 600 r, and at ages ranging from 19 to 243 hours.

The results are as follows:

(1) In 45 per cent ot the cases there was little or no development after irradiation.

(2) In 24 per cent of the cases, chicks died in the shell at full development.

(3) In 11.9 per cent of the cases, chicks died after development had proceded for several days (15 ± days).

(4) In 9 per cent of the cases, chicks' lower extremities were paralyzed.

(5) In 6 per cent of the cases, the inferior extremities were deformed. The deformity varying from a distortion of the toes to a total absence of one or both feet.

(6) In 1.4 per cent of the specimens, there was a hydrocephalic head deformity.

(7) One per cent of the cases were miscellaneous deformities.

(8) No appreciable difference in the results were noticed between chicks irradiated with shell and shell-membranes opened and those with shell intact.

(9) Eggshell of ordinary density with its membranes absorb 0.2 r of x-rays per minute.

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X-RAYS AND RADIUM IN THE TREATMENT OF TUMORS OF THE CONJUNCTIVA[1]

By DR. GUSTAV PETER, American Hospital, *Mexico City*

IN a former paper, published in "Revista de Radiologia y Fisioterapia," 1935, and based on 120 cases of cutaneous epithelioma and 12 cases of epithelioma of the conjunctiva, treated with x-rays of different wave lengths, gamma rays, and beta rays, I presented the following conclusions:

(1) In working with oscillatory electromagnetic waves of from 1.0 to 0.16 Ångstrom units for the destruction in general practice of cutaneous epithelioma, no optimum wave length was found, the use of which could be preferred to that of others of the series. In other words, the same results may be obtained with x-rays as with radium under the specified conditions.

(2) On the other hand, in comparing oscillatory electromagnetic rays with beta corpuscular rays, the conclusion is reached that in all this group of radiations, including the moderately penetrating gamma rays in the middle of the gamma-ray scale, the beta rays, which are the least used in present-day practice, are the most active.

More recently I have had the opportunity to test the practical value of these conclusions due to the following circumstances:

In a case of epithelioma of the conjunctiva, cured 14 years ago with x-rays of 0.2 Ångstrom unit, a glaucoma developed a few months after the irradiation. Since then I have used a capsule of beta rays (10 milligrams) and a 0.2 mm. silver filter in all ophthalmic cases, with the result that no complications have followed any of these treatments. It is supposed that the small penetration of the beta rays has allowed such satisfactory results without complications involving deeper tissues, such as the ciliary body and the crystalline lens.

A *B* *C*

Fig. 1-*A*. Case 1, Group I (see table), showing condition on June 1, 1934; Fig. 1-*B*, on June 12, 1934; Fig. 1-*C*., on Aug. 15, 1934.

Two years ago, on checking the impermeability of my radium containers, I found that the capsule had a microscopic defect, impossible to locate exactly, which allowed the emanation to escape. In order to correct this, I was obliged to seal hermetically the entire capsule in a 0.3 mm. silver filter. This resulted in the disadvantage that the filter absorbed nearly all the beta rays, and the intensity was thereby reduced from 15 r to 3.2 r per minute.

Figs. 1-*A* to 2-*B*, inclusive, show two of the 12 cases of conjunctival epithelioma cured with the original capsule. The case shown in Figures 3-*A* and 3-*C* was cured with the capsule after it was hermetically sealed in the silver filter.

On comparing the details of procedure in

[1] Translated from the Spanish by Mrs. Julia Baker.

Groups I and II, it will be noted that the dosage in Group II is three and a half times that in Group I. Furthermore, three months of almost daily applications were necessary for complete cure, and the patient complained of pain five months after the beginning of the treatment, in spite of the fact that, clinically, the eye was normal. No other patient has suffered prolonged discomfort.

The fact that the total dosage as well as the total duration had to be increased in using Group II technic is explained by the circumstance that the intensity and the daily fraction was four and a half times less in that group.

In both groups an exudative epidermitis developed on the edges of the eyelid in the areas not entirely protected by lead. Using Group II technic, the epidermitis developed in a more pronounced form even before the tumor had disappeared com-

A *B*

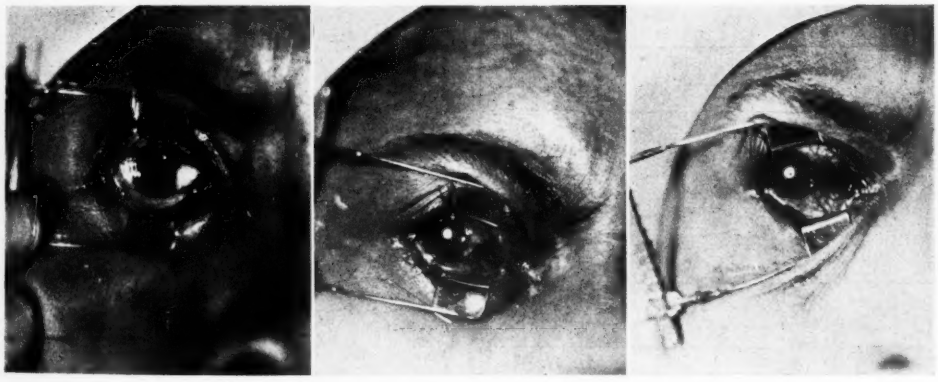
Fig. 2-*A*. Case 2, Group I (see table), showing condition on May 15, 1933; Fig. 2-*B*., on Aug. 1, 1935.

A *B* *C*

Fig. 3-*A*. Case 1, Group II (see table), showing condition on March 1, 1935; Fig. 3-*B*., on June 24, 1935; Fig. 3-*C*., on June 15, 1935.

A *B* *C*

Fig. 4-*A*. Case 1, Group III (see table), showing condition on June 20, 1935; Fig. 4-*B*., on July 2, 1935; Fig. 4-*C*., on Aug. 7, 1935.



A *B* *C*

Fig. 5-*A*. Case 2, Group III (see table), showing condition on July 28, 1935; Fig. 5-*B*., on Aug. 11 1935; Fig. 5-*C*., on Sept. 11, 1935.

pletely, and lasted a week longer. At the present time (the last of November, 1935), the eye is clinically normal, but the patient still complained of pain in the eyeball during the last week of August.

The increased amount of work for the physician, as well as the trouble for the patient using Group II technic, prompted me to look for another solution of the problem. I found this by applying the first conclusion mentioned in this paper, *i.e.*, the same results may be obtained by the use of either x-rays or radium.

The two cases of Group III were, there-

TABLE[1] I

	Ma.	Kv.	Half value layer	Filter	Distance	Field	Pause between fractions	r/a per fraction (r/a = international r units measured in air)	Time per fraction	r/a per minute	Number of fractions	Total doses per field	Total time for tumor	Total time for series	Number of series	½ value layer med.	Reaction	Reaction time
Group I Capsule Radium Beta 10 mg. Fractional Irradiation			(calc.) 0.01 Cu equivalent to λ 0.8 Å. Al 0.4 mm.	0.2 Ag + 01 monel	0.5 cm.	1 × 1.5 cm.	24 hr.	150 or 525	10 min. or 35 min.	15	7 2	1000 r/a	70 min.	7 days 2 days	1 1	0.32 brass	Exudative epidermitis: erythematous conjunctivitis Duration, 1 week	Epidermitis began 14 days after first treatment: cured in 4 weeks
Group II Capsule Radium B 10 mg. Protracted Fractional Irradiation			measured 0.052 Cu equivalent to λ 0.43 Å. Al 1.75 mm.	0.5 Ag + 0.1 monel	0.5 cm	1.0 × 1.5 cm.	24 hr.	48 and 64 190 570	15 min. + 20 min. + 1 hr. + 3 hr.	3.2	16 +39 +8 +1 64	3600 r/a	31 hr.	3 months	1		Exudative epidermitis: erythematous conjunctivitis Duration, 1 week	Began after 3 months: cured in 3 months
Group III X-ray Fractional Irradiation	2.5	44	measured 0.011 Cu equivalent to λ 0.8 Å. Al 0.4 mm.	no filter	25 cm.	1.5 × 2 cm.	24 hr.	150 r/a	10 min.	15	12	1800 r/a	120 min.	2 weeks	1		Exudative epidermitis: erythematous conjunctivitis Duration, 1 week	Began after 14 days: cured in 4 weeks.

[1] These cases have been kindly referred to me by Dr. Rafael Silva, Dr. Torres, and Dr. Teaza, Directors of the Ophthalmological Hospital de la Luz, Mexico.

fore, treated with x-rays and cured. The fractional dosage, intensity, field, and half value layer were reproduced as accurately as possible.

After a series of treatments of 12 fractions a day (except Sunday), the tumor presented the typical aspect of destruction, which is characteristic of radium irradiation treatment. A week later, the destruction was almost complete and a slight irritation of the skin had developed on the border of the eyelid in unprotected spots. A week later, both the tumor and the irritation of the skin had disappeared. The course of the disease and the character of the exudative epidermitis were the same as when using Group I technic, and experience of many years has proved that the eyeball as well as the skin can easily stand this treatment.

The technic used in Group III represents a special case of fractional technic, the relatively harmless effects of which on the skin, using shorter wave lengths, have been carefully studied and are well known (Miescher, Borak, and others). Since we know that the dangers of certain dosages of given intensities diminish with the increase in wave length, it is not to be supposed that the exudative dermatitis produced with Group III technic would be more harmful than the epidermitis which we apply without hesitation to cutaneous epithelioma, in spite of the fact that penetration there is greater and the field larger. In epithelioma, we know that this technic is easily tolerated by the skin and that it produces no detrimental after effects.

The similarity of the technic used in Groups I and III permits the hypothesis that, for the ocular bulb, the ciliary body, and the crystalline lens, especially, the effect is the same.

On comparing the technic of Groups I and III, it is noted that the total dosage with x-rays had to be greater than the dosage with radium (x-rays, 1,800 r—radium, 1,000 r). In addition to this, two weeks were needed for the x-ray treatment, whereas the same result was accomplished with radium in half that time. This fact

supports the second conclusion mentioned above, *i.e.*, that the corpuscular rays are more active than the oscillatory rays.

These clinical observations naturally must be corroborated by exact physical methods. The ionization chamber of the Mekapion, with which the measurements were made, is guaranteed for wave lengths of from 70 to 220 kilovolts. The x-rays used were 44 kv., corresponding to a half value layer of 0.01 copper (0.8 Ångstrom unit). The half value layer of the radium capsule before its repair had only been calculated at 0.01 copper. The direct measurement at the important moment of discovering the defect was not possible, due to the danger of re-charging the ionization chamber with emanation. For these reasons, one cannot overlook the possibility that the smaller total dose necessary in the radium treatment may be explained by less penetration than that calculated, or in other words, by more absorption in the wall of the ionization chamber.

CONCLUSION

Epithelioma of the conjunctiva may be cured with x-rays as well as with radium, by using a technic which differs only in minor details of small, practical, or economic importance.

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EDITORIAL

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INFORMATION CONCERNING THE NATIONAL HOUSING ACT AS IT MAY AFFECT RADIOLOGISTS

The National Housing Act which is being administered by the Federal Housing Administration was designed to be of help to the professional and scientific man as well as the layman, the small home owner, the man in business and industry.

The regulations under which it is easy to get insured modernization credit are so attractive that the radiologist, or other specialist whose establishment may be in need of modernization, repair, or new equipment essential to the conduct of his practice, should be familiar with the provisions.

First it should be explained that it is possible to get insured credit for as much as $50,000, or for a few hundred, depending upon the need and the credit standing of the borrower. The carrying charges may never be in excess of the equivalent of a $5 discount for each $100 face value of a one-year monthly installment note. The loan is to be repaid in equal monthly installments and may be spread over a period of as long as five years.

The Federal Housing Administration does not lend the money. The transaction is made through ordinary banking channels with banks which have entered into contracts with the Federal Housing Administration, under which the administration insures them against loss up to 20 per cent of the total of such loans. There are one or more such institutions in practically every community in the United States.

The arrangement is so attractive that banks ordinarily are more than willing to extend the accommodation and it is now easy to get the required credit in instances in which it has been difficult if not impossible during the period of the depression.

The essential requirements are that the lending agency must consider the applicant a good credit risk and must be satisfied as to security in instances in which security is considered advisable. This is because the banker instead of the government makes the loan.

The question of the type of equipment which would be considered eligible for a loan may in many instances be answered by the dealer or his representative. Broadly, it would include the installation of non-portable and durable character—sufficiently so to outlive the period of the loan.

Rulings have already been made that the following are eligible for loans:

X-ray machines, x-ray generators, fluoroscopes, certain types of deep therapy equipment, certain types of x-ray dosage measuring equipment, x-ray tilt tables, horizontal x-ray tables. Equipment of this type would ordinarily be considered eligible.

In instances in which there is any doubt as to eligibility, suggest to the dealer that he send a complete description, with photographs, to the Federal Housing Administration, Washington, D. C., for an official ruling.

Scientists or professional men who maintain their offices in residences are limited to a loan of $2,000. Those who have offices in office or apartment buildings may obtain larger sums, if the banker approves.

Medical men who maintain jointly a clinic or diagnostic center may find it desirable and profitable to modernize and repair the structure (loans for this purpose are usually eligible) and to install the latest devices essential to modern practices.

The legislation, as explained, took this into consideration when it included hospitals in the list of properties upon which large loans may be made.

Another and final point is that the loan is in cash for the full amount, so that purchases and repairs or modernization may be made on a cash basis with the benefit of the cash discount.

IN MEMORIAM

BUNDY ALLEN, M.D.

When members of the Radiological Society of North America gathered for their 1935 Annual Meeting, they were shocked and saddened to hear of the death, a few days before, of their

friend and past president, Bundy Allen. He was the victim of a collision between his car, driven by himself, and a bus, the latter being used by a party returning from a fishing trip.

The late Bundy Allen, M.D.

Dr. Allen lived more than twenty-four hours, but did not regain consciousness; the skull fracture which he suffered was of so serious a nature that the physicians in attendance knew that the chance of his recovery was slight. His eleven-year-old son, Joseph, who was in the car with him when the bus struck his car, was not seriously hurt.

Bundy Allen was born Aug. 21, 1885, at Carbondale, Illinois, in which town he received his elementary schooling. He took a pre-medical course at the University of Indiana and received his M.D. degree in 1912 from St. Louis University, at St. Louis, Missouri. After practising medicine for two years in that city, he went to the University of Iowa, at Iowa City, where he was head of the Department of Roentgenology for eleven years. He married Miss Edith Cochenour the same year he took his degree in medicine. In 1926 Dr. Allen and his

family moved to Tampa, Florida, where he confined his practice of medicine to the specialties of roentgen ray and radium work.

Dr. Allen was a member of his local, state, and national medical societies, the Radiological Society of North America (charter member), the American Roentgen Ray Society, the College of Radiology, and the British Institute of Radiology, of England. He was formerly Associate Editor of RADIOLOGY. His active interest was manifested in civic associations and social welfare organizations in his home city.

His loss is felt keenly by his friends, and the grief felt over his untimely death is deep and genuine.

HARRY B. PODLASKY, M.D.

Harry B. Podlasky, M.D., died Sunday afternoon, Nov. 3, 1935, of coronary thrombosis. The attack was entirely unexpected since he had never had any symptoms of coronary artery disease. He was 52 years of age. For his intimates, his death was a shocking calamity, and by his many friends throughout this country his passing was deeply felt. He is survived by his wife and daughter, who have no small comfort in the tokens of kindly and sympathetic appreciation from an almost unsuspected host of friends.

The late Harry B. Podlasky, M.D.

"Pod," as he was affectionately known, did not leave a great monument of medical achievement in the sense of accomplished research or extensive bibliography. He was always active, however, in radiological circles, and his handsome figure and vigorous personality lent color and dignity to medical gatherings. He was a member of both of the great national radiological societies, and recently had become a Fellow of the American College of Radiology and a diplomate of the American Board of Radiology. In Wisconsin, his activities in the State Section of Radiology, Milwaukee Roentgen Ray Society, and Milwaukee County Medical Society were always in the interests of the profession. He founded and directed the Department of Radiology at Mount Sinai Hospital, Milwaukee. Into Mount Sinai Hospital went energy, enthusiasm, and devotion of a kind and degree not given to any of his other interests. For him, this hospital loomed as the hub of his medical community, and he visualized it as a source of energizing medical training and education for the novitiates in medicine.

He fulfilled the destinies of the true physician by his activities as a teacher, being head of the Department of Radiology at Marquette University and Editor of the section on x-ray of the "Wisconsin State Medical Journal."

As a radiologist, his *forte* was in the field of diagnosis, and it may certainly be said that he possessed not only diagnostic ability of a superlative degree, but, coupled with that, knowledge of medical practice inherited from his years of general medicine before specializing in radiology. He strongly felt the need for radiologists to have at their command a broad concept of medicine and sound training in pathology and physiology. He frequently laid the shortcomings of specialists in his field to the lack of a broad medical education. His friends frequently criticized him for his almost ultraconservative readings of diagnostic investigations. Of late years he was prone to call attention to the tendency for medicine to develop away from the arts and to adopt methods of accuracy, and in that regard, radiology in his opinion needed scientific control, and he much deplored the abuses of both the science and the practitioners of radiology by commercializing and economic influences. He was a vigorous protagonist of the development of the specialty under national advisement, and strongly felt that the difficulties and problems besetting this division of medicine could be solved satisfac-

torily only by national organization. He was among that small group who were willing to accept the brunt of sacrifice in the defense of a principle. A vigorous champion of personal and group rights, radiologists have lost in him a man of idealism and practical common sense. For him, medicine was a religion. Ethics, right living, and right behaving had only one meaning. In his teaching, it was not enough for him to teach subject matter, but always he had in mind the teaching of the conduct of practice to students and recent graduates. He exalted the profession of medicine. Jefferson Medical College, from which he was graduated in 1909, may well be proud of Harry Bernard Podlasky.

NORBERT ENZER, M.D.

ANNOUNCEMENT

Harper & Brothers announce that they have acquired the medical book publishing business of Paul B. Hoeber, Inc. A program of expansion is planned, and medical books will be published under the imprint of Paul B. Hoeber, Inc., Medical Book Department of Harper & Brothers. Mr. Paul B. Hoeber, the founder, remains in charge of this department.

Paul B. Hoeber, Inc., was established in 1901 as a medical bookstore. The first book published under the Hoeber imprint—Ehrlich's "Specific Therapeutics"—appeared in 1909. Since then it has pioneered in the publication of authoritative medical books which have been noteworthy also for their fine manufacture. The firm is well known for its series of Medical Monographs, which includes works by Francis R. Packard, Hugh H. Young, David Riesman, Sir Humphry Rolleston, Frederick Tilney, Charles A. Elsberg, and other authorities. It has specialized in medical history, and established in 1917 the "Annals of Medical History," internationally known as the outstanding publication of its kind. Among the eminent books which have appeared under the Hoeber imprint are "The Brain from Ape to Man" by Frederick Tilney, "Nervous Indigestion" by Walter C. Alvarez, "Pediatrics of the Past" by John Rurah, and the "Annals of Roentgenology" of which sixteen volumes have been published to date.

BOOK REVIEWS

SYMPOSIUM ON SILICOSIS. An Unofficial Transcript of the Silicosis Symposium Held in Connection with the Trudeau School of Tuberculosis at Saranac Lake, New York, June 18 to 22, 1934. Edited by B. E. Kuechle, Claims Manager, Employers Mutuals, Wausau, Wisconsin. Introduction by Dr. Leroy U. Gardner. Paper-bound volume of 99 pages and no illustrations. Published by The Storey-Bellack Company, Inc., Wausau, Wisconsin, 1934. Price: $5.00.

The book containing the unofficial transcript of lecture notes taken during the Silicosis Symposium at Saranac in June, 1934, is a small one but of great value. Each lecturer approved the notes of his lecture before it went to press. It is important to note that the financial returns are being given to the laboratory at Saranac for research on silicosis.

The form is that of abstracts of the papers given at the conference, and the discussion of those papers. It is unusually well written, very brief, and very readable. The contributors to the conference were men of ability who had real contributions to make. As a result of these contributions, the various types of silicosis can be recognized, the progress of the disease understood, the treatment improved, and its prevention made possible.

On the whole, this book is the most constructive piece of work on silicosis that has ever been attempted and brought to a conclusion. It should be read and studied by every radiologist, every clinician interested in diseases of the chest, and every physician who comes in contact with industrial medicine.

DIE KLINISCHE RÖNTGENDIAGNOSTIK DER INNEREN ERKRANKUNGEN. By DR. HERBERT ASSMANN, Professor and Director of the Medical Clinic and of the University of Königsberg. Fifth Edition. With 1248 pages, 1216 illustrations, and 10 tables. Parts I and II. Published by Julius Springer, Berlin, 1934. Price: Rm. 87; bound, Rm. 95.

This well-known text-book, which has served for instruction and reference in the hands of many young and old radiologists and physicians all over the world, has been reprinted in its fifth edition. The author, who is a Professor of Internal Medicine at Königsberg, Germany,

has succeeded admirably in presenting that field of roentgenology which concerns internal medicine. For the radiologist it is of particular interest to see in what way a man, whose specialty is internal medicine, uses the roentgen rays, the approach being clinical primarily, and roentgenological secondarily. For the general diagnostician these two volumes, with 1248 pages, represent a connecting link between medicine and roentgenology; to read this book might be easier for him than to study a book of roentgenology written by a radiologist.

Volume I (466 pages) contains the chapter on the heart and blood vessels, mediastinum, lungs, pleura, and diaphragms. The normal roentgen appearance and the question of determining actual heart-size are related in detail. Pathological changes in size and contour are discussed at great length. The recent, much-talked-of kymography is mentioned, and some newly described roentgen symptoms, valuable in the diagnosis of heart diseases, are quoted, such as aneurysms of the heart, tumors of the heart, and calcification of the valves. The aorta and its roentgenologically demonstrable diseases are allocated 30 pages and not much more could be said to complete that subject. Arteriography takes up only a few lines.

The mediastinum and its pathology is dealt with adequately but probably not as extensively as the material on the heart.

The treatise on the lungs, pleura, and diaphragms might constitute a book in itself. One misses the mention of a vena azygos lobe in the lower lung-fields, which has been described recently. It is interesting to note that massive atelectasis is related only by quoting the literature, and that the reproductions of two chest films, showing this condition, are taken from Sante's book, "The Chest." It would appear from this that such a condition is not commonly recognized in Europe, for it is certainly not rare in the United States. Many pages are devoted to a fully sufficient description of the roentgen signs of all stages of tuberculosis. However high the author's regard is for the outstanding importance of roentgenography, he still insists on the extensive use of all clinical diagnostic means, especially for the question of activity and prognosis. Unusual and rare diseases of the lungs are mentioned, but their description is hardly detailed enough to make this section useful as a reference. All one wants to know about the radiological features of diseases of the pleura and diaphragm will be found in these respective chapters.

Volume II deals extensively with diseases of the gastro-intestinal tract (325 pages), the other abdominal organs, the urinary tract, the nervous system, bones, joints, and muscles.

The roentgen examination of the esophagus is treated proportionately. It is interesting to note that the author calls the diagnosis of gastric diseases by means of multiple films, the American method, while the preference for fluoroscopy is called the Vienna method. Apparently, he does not know that the procedure of Cole is not generally followed at all throughout the United States, and that many American examiners put a higher valuation on fluoroscopy than on films. The author, himself, prefers a combination of both. Considerable attention is paid to functional changes of the stomach. Organic diseases are examined for the roentgenological equivalent of the pathological features mainly, and so-called secondary signs are considered of only secondary importance. Cholecystography receives the description which it deserves. A clinical syndrome of ptosis and atony of the gall bladder is held doubtful, but it is stated that adhesions may often be demonstrated, especially when the gastro-intestinal tract is filled by barium.

Methods used in the examination of the urinary tract are presented in a way which suffices for the field of internal medicine.

Roentgenology of the skull (including a special chapter on encephalography) gives the reader a general outline of the value of this method. It is mentioned that in exceedingly rare instances, spinal cord tumors might lead to roentgenologically demonstrable erosions of the vertebræ. The important contribution of Camp, of the Mayo Clinic, who found this true in a high percentage of cases, is not quoted.

Only those diseases of bones and joints that concern internal medicine are described; and yet, the chapter contains about 140 pages.

It seems an almost unbelievable achievement for one man to master the entire field in such a remarkable way. Certainly the character of such a text-book is more individualistic than one containing contributions of several authors. The German literature is quoted extensively, while the literature in other languages is related very sparsely and makes up less than 5 per cent of all quotations. Thus, some very important American contributions have been missed.

For any physician who specializes in roentgenology or internal medicine and who reads the German language, this work is to be recommended as highly as ever. It is the modernized Assmann, truly bigger and better.

THE 1935 YEAR BOOK OF RADIOLOGY. *Diagnosis*, edited by CHARLES A. WATERS, M.D., Associate in Roentgenology, Johns Hopkins University; Assistant Visiting Roentgenologist, Johns Hopkins Hospital. Associate Editor: WHITMER B. FIROR, M.D., Assistant in Roentgenology, Johns Hopkins Hospital. *Therapeutics*, edited by IRA I. KAPLAN, B.Sc., M.D., Director, Division of Cancer, Department of Hospitals, City of New York; Director, Radiation Therapy Department, Bellevue Hospital, New York City; Director, New York City and Brooklyn Cancer Institutes; Associate Radiologist, Lenox Hill Hospital, New York City; Clinical Professor of Surgery, New York University Medical College. With 580 pages and 619 illustrations. Published by The Year Book Publishers, Inc., Chicago, Illinois, 1935. Price: $4.50.

This annual review of the current radiological literature in its short period of existence has so established itself that its appearance each year provokes the same satisfaction as the coming of a favorite magazine. Notwithstanding the objections to short-cuts to knowledge, no individual who is seriously interested in the advancement of radiology can peruse this year book without profiting much thereby and deriving an added stimulus for his work.

A review of the diagnostic section of this volume emphasizes even better than its predecessors that this branch of radiology is living in the "injection era" and with the progress that has been made to date with radiopaque substances, future revelations are interesting to speculate about. Saitos' unique work on the visualization of peripheral nerves and their pathologic changes (neurography) is an excellent example of what may be expected.

Although no unusual developments in radiation therapy occurred during the year the literature concerning this phase of radiology was profuse. It is evident that this branch of radiology is suffering from voltage "growing pains." "It is, however, a moot question whether x-rays generated by superpowered apparatus have greater therapeutic potency than those of the usual kind, generated by the 200 kv. apparatus." Wright's lecture on the value of pre-operative radiation for breast cancer and Crossen's declarations that "radiation is the treatment of choice for carcinoma of the cervix" and that "surgery should supplement, not displace, this more desirable procedure" are two contributions of note.

An adequate review of the entire volume is obviously impossible because of its nature. Needless to say the authors have condensed the current radiologic literature in a manner that should insure the popularity of this work. The illustrations are profuse and excellently reproduced.

THE DOCTOR'S BILL. By HUGH CABOT. With an introduction by A. Lawrence Lowell. A volume of 313 pages and no illustrations. Published by Columbia University Press, New York, 1935. Price: $3.00.

The title of this book sounds far more popular than the contents will be to the average physician who is trying to collect his bills. This is a philosophic treatise upon the trends in methods and practice. It is quite different in its viewpoint from the book by Christie (Economic Problems in Medicine).

The medical profession recognizes Hugh Cabot as an illustrious medical representative, administrator, and urologic surgeon. The name of Cabot is great in medicine, whatever direction is pursued. The introduction by A. Lawrence Lowell, formerly president of Harvard University, is a further guarantee that the conservative, God-communicating sequence has been preserved.

The title is somewhat misleading because the substance of the book takes a far look into the trends of medical practice. The viewpoint is more philosophical and detached than practical. One should not say that it is impractical but certainly the book offers little comfort to those of us who are harassed by the economic problems of the moment. It finds flaws rather than faults with the activities of the Bureau of Economics of the American Medical Association. The book is not anti-social or anti-American Medical Association. It is just a cold analysis of medical trends and Cabot seems to believe that things are going to be different—in fact, quite different—as the inevitable turning-tables of social organization are applied to medical practice.

Cabot quotes time and again from Sir Arthur Newsholme, the eminent English public health protagonist. Sir Arthur has a good reputation but his American debut and subsequent ex-

periences were devised by the lately developed Kingsbury of the Milbank Fund. Unquestionably this has interfered with the proper reception of Sir Arthur's ideas by the American profession. No less an individual than the great Welch, of Hopkins, was sponsor, partisan, and friend of Sir Arthur. But whatever influence Sir Arthur's visit had on America, he has failed to provoke any echo or chorus of praise by organized medicine here.

Of course the average American physician looks no farther than the end of his nose for the solution of an economic problem. Therefore, in choosing the accessible hay of the moment he may be missing the great pasture of perpetual nourishment. In spite of the resoluting of county, state, and national organizations in medicine, there are inevitable changes that are now in the process of sedimentation. The waters are stirred unreasonably and beyond all ocular vision of immediate solution. The dough is in the kneading, the leaven cannot be measured. How far are the present activities in so-called social security going? Will they engulf the established methods of individualistic and collective group methods of practice? Will they demand the regimentation of medicine to the extent that police protection, public education, water supply, and fire protection now enjoy compulsory taxation?

Cabot's concluding chapter is entitled, "Where do we go from here?" The theme of his message is "he that pays the piper, calls the tune." Therefore, the taxpayer gets far more house from Cabot than the theme songs of 1934–1935 American Medical Association meetings grant. Cabot feels that in meeting the demands of science, medical organization has progressed to an adequate extent, but that there has been failure to recognize the problem of social medicine; therefore, the profession is now suffering from the folly of its own neglect. Cabot constantly reiterates his concern for the public interest. He believes that physicians should be paid for their care of indigent and semi-indigent patients by state payments, taxation and by collective subsidies for workman's compensation industrials, poor geographical population groupings, etc. Cabot does not fear, but rather encourages, the introduction of laymen into medical groupings, boards, committees, etc., to provide stability (in finances) and register public opinion. He favors more and better public health personnel, nurses and social workers and a larger active participation by physicians.

Cabot does not argue for state medicine. Rather, does he analyze the inadequacies of medical organization toward what he considers the inevitable demands of public interest. Therefore this may be considered as the viewpoint of a physician whose family training and life have been ultra-conservative, and that it records his reactions to the clamor of propaganda and foundations and to the alleged stubbornness or short-sightedness of organized medicine. Cabot discusses the trends toward social security and the wider distribution of the benefits of invention, research, and what we are now calling civilization. It is not an antidote to Christie's book. Rather, is it a bitter pill, and here's hoping that the profession is not jaundiced and don't need it. But it is written by a Cabot and you cannot just rub it off your list.

RADIUM TREATMENT OF SKIN DISEASES, NEWGROWTHS, DISEASES OF THE EYES AND TONSILS. By FRANCIS H. WILLIAMS, M.D. (Harv.), S.B. Massachusetts Institute of Technology; Senior Physician Boston City Hospital; Fellow American Academy of Arts and Sciences; Emeritus Member Association American Physicians; Member Société de Radiologie Médicale de France; Corresponding Member K. K. Gesellschaft der Aerzte in Wien; Honorary Member American Radium Society, American Roentgen Society, Radiological Society of North America, etc. Author: "The Roentgen Rays in Medicine and Surgery" (3 editions), 1910–1903. Bound volume of 118 pages, 12 figures. Published by the Stratford Company, Boston, Mass., 1935. Price: $2.00

This is a small, concise volume. The text is presented in an interesting manner, interspersed with many case reports and photographs of instruments designed by the author for the radium treatment of eye and throat lesions. The ophthalmologist and laryngologist will find several valuable therapeutic suggestions for problems in their domain. The radiologist, however, will especially esteem the book for its historical and inspirational value. The author is one of the true pioneers of radiology and describes many of his early researches and methods of measurement. His enthusiasm for radium therapy after thirty years of experience is a testimonial to the value of radium in medicine and is also an equal testimonial to the painstaking and conscientious life work of the author.

LEISTUNG UND GRENZEN DES RÖNTGENVERFAHRENS BEI DER ERKENNUNG TUBERCULÖSER LUNGENVERÄNDERUNGEN (The Accomplishments and Limitations of the Roentgen Method in the Study of Tuberculous Lung Changes). A comparative roentgenologic, clinical, and anatomic study. By Priv.-Doz. DR. WALTER SCHMIDT, Director and chief physician of the Tuberculosis Hospital, Heidelberg-Rohrbach. With the collaboration of Priv.-Doz. DR. H. WURM, Prosector, Pathologic Institute, Heidelberg. A volume of 80 pages and 104 illustrations. Published by Johann Ambrosius Barth, Leipzig, 1935. Price: Rm. 10.

This small monograph, with only 26 pages of text but with 104 excellent illustrations, presents a correlation of the roentgen and autopsy findings in a series of 170 well-studied cases of pulmonary tuberculosis. It is Schmidt's purpose to supplement the original work of Graf and Küpferle, in this same field, by bringing to it the knowledge of tuberculosis gained during the past decade, and particularly to add studies of the early adult lesion which were not covered in their monograph.

The introduction by Dr. Wurm details the method of preparation and study of the pathological specimens and their comparison with the roentgenograms made during life. By this means the authors are enabled to present authoritative data on the significance of the roentgenographic findings in a variety of tuberculous processes.

The discussion includes detailed comment on the following: old childhood infections, the early adult lesion, the question of endogenous or exogenous origin of adult lesions, the possibility of the roentgen diagnosis of various pathological states, the recognition of the early cavity, the findings in old cavities, the significance of the roentgenographic findings in prognosis, the importance of proper roentgen technic.

There is little in this monograph that is unfamiliar to the American roentgenologist or phthisiologist. Nevertheless, there is presented abundant confirmation of many relatively new ideas which in itself makes the book worthwhile. The material is presented most concisely so that there is a great deal of meat in the few pages of text. Particular attention may be called to the author's classification of tuberculous lesions and to the detailed discussion of the early adult tuberculous process (*frühinfiltrat*). The data on the development, diagnosis, and differentiation of early tuberculous cavities are also of unusual interest. The statements on the value of roentgenoscopy, x-ray paper, stereoscopy, and serial roentgenography are of considerable value, although many of the author's conclusions will not be generally accepted.

THE RADIOLOGY OF BONES AND JOINTS. By JAMES F. BRAILSFORD, M.D. (B'ham), M.R.C.S. (Eng.), Radiological Demonstrator in Living Anatomy, the University of Birmingham; Honorary Radiologist to the Queen's Hospital, Birmingham; Honorary Radiologist to the Royal Cripples' Hospital and the Warwickshire Orthopedic Hospital; Radiologist to St. Chad's Hospital and the City of Birmingham Infant Welfare Centers; Late Radiologist, the Birmingham War Hospitals and Ministry of Pension Hospitals. Fabrikoid binding; 500 pages and 310 illustrations. Published by William Wood and Company, Baltimore, 1934. Price: $9.00.

Extensive radiologic experience, knowledge of clinical medicine, and a keen appreciation of relative values are apparent upon every page of this beautifully printed and excellently illustrated volume. Furthermore, the index is splendid and this is always appreciated by those who consult a book for help upon both the usual and unusual in roentgen practice. A good index does not always secure the praise deserved, but a bad index can banish an otherwise useful book to oblivion. Even the table of contents is extensive and valuable in Mr. Brailsford's book.

The author devotes an ample chapter to the discussion of each of the ordinary anatomical divisions and joints of the skeleton, lists and discusses the usual deformities, congenital and acquired, together with the pathological displays which are characteristic. The sense of values of the author is apparent when he devotes four chapters to the spine, its deformities, congenital variations, and pathological vagaries.

The author has certainly completed his ambition to describe and illustrate bone changes in health, deformity, and disease, to indicate the significance of the roentgen evidence and to record the advances in roentgenology that up to this time have been present only in the journal literature.

The reviewer can remember picking up this book at a publishers' exhibit and then replacing it upon the rack as just another book on roentgen interpretation. But, upon looking through it for reviewing purposes, he was so impressed with the completeness of the roentgen information that he found himself reading whole chapters. In an attempt to discover some weakness he could discover only an absence of parathyroid calcium deficiency discussion. This is, however, not a real weakness because the subject is not settled; it is only muddled by the

inadequacy of final information and clinical support of the original contentions of early contributors. So Mr. Brailsford is probably correct in omitting this controversial subject.

If any practising roentgenologist possesses Koehler's volume upon the borderline of the normal and abnormal in "Roentgenology" and Geschickter and Copeland's tome on "Bone Tumors" and now adds Brailsford's "Radiology of Bones and Joints," he will have completed his library for bone displays upon the roentgen-ray film. Of course, to-morrow may provide another and all-comprehensive volume, but until to-morrow comes let us rest content with the possession of this excellent book.

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H. A. JARRE, M.D., of Detroit, Mich.
ERNST A. POHLE, M.D., Ph.D., of Madison, Wisc.
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APPARATUS

Tomography II (Theoretical Considerations). G. Grossmann. Fortschr. a. d. Geb. d. Röntgenstrahlen, 1935, **51,** 191–208.

Geometric and physical deliberations of the various technical and practical tomographic propositions. This paper should be read in the original by those interested in the subject.

H. A. JARRE, M.D.

CANCER (THERAPY)

Cancer of the Cheek (Buccal Mucosa): Study of 99 Cases, with Results of Treatment at the End of Five Years. Hayes E. Martin and Otto H. Pflueger. Arch. Surg., May, 1935, **30,** 731–747. (Reprinted by permission from British Med. Jour., Sept. 21, 1935, p. 44 of Epitome of Current Medical Literature.)

The authors review 99 cases of carcinoma of the cheek which occurred in the five-year period from 1925 to 1929. The series included all cases, however advanced, in which there was histologic proof of the presence of cancer, and the end-results are based on the histories of patients observed for five years or longer.

Cancer of the cheek is chiefly a disease of old age, and is nine times as frequent among men as among women. The mid-portion of the cheek, opposite the occlusal level of the teeth, is the most common site, although the disease may appear anywhere on the buccal mucosa or in either of the buccogingival grooves. Direct local extension of the primary growth into the adjacent soft parts may take place at an early stage of the disease, and was seen in over 60 per cent of cases. Chronic irritation to the buccal mucosa by sharp or broken teeth, ill-fitting dental appliances, syphilis, and tobacco is the most obvious etiologic factor in carcinoma of the cheek. There was evidence of dental irritation in 23 cases. Leukoplakia was found in about 70 per cent of cases.

Cancer of the cheek is found to be epidermoid carcinoma in 95 per cent of cases. The disease is symptomless in the early stages, and appears at first as a small, ulcerated, indurated mass, which may or may not protrude above the surface of the mucosa. Unless the lesion begins in the center of the cheek the tendency is toward an early invasion of the upper or lower jaw, the lateral pharyngeal wall, or the palate. As the tumor enlarges it becomes fissured and necrotic, infection takes place, followed by swelling of the cheek and tenderness or pain. Metastasis, usually to the submaxillary lymph nodes, occurs comparatively late in the course of the disease.

The most successful treatment was found to be a combination of irradiation and surgical intervention. Of the group under review, 28 patients have been free from disease for from five to eight years.

The Relative Value of Surgery, Radium and Roentgen Therapy in Carcinoma of the Breast. Isaac Levin. Am. Jour. Roentgenol. and Rad. Ther., January, 1935, **33,** 59–70.

Surgery, radium and roentgen therapy are the only methods in our possession for treating cancer of the breast. The modern methods of radical surgery in cancer of the breast represent a great advance over the old methods. However, even with the latest methods of surgery not more than 10 per cent of all patients remain well five years after operation.

In view of the small percentage of the successful cases, it is advisable not to differentiate so severely between radical and palliative surgery; on the one hand, widen the indications for surgery and, on the other, use less extensive methods of operating. Surgery should not be considered as the only therapeutic method, but, rather, as an indispensable adjuvant to radiotherapy. Electrosurgery may improve the surgical results.

Radiotherapy is the nearest to a specific remedy we possess in cancer and is, from this standpoint, superior to surgery and has a wider applicability. Limitations of radiotherapy are of such a nature as to be frequently overcome by a correct co-ordination with surgery.

S. M. ATKINS, M.D.

CARBUNCLES

Roentgen Treatment of Carbuncles. Whitmer B. Firor. Am. Jour. Roentgenol. and Rad. Ther., January, 1935, **33,** 71–74.

In the past three years the author has treated 56 carbuncles with x-ray therapy and produced, like many other roentgenologists, brilliant results. He deplores the surgeons' and physicians' failure to use this method of treatment more frequently and believes their apathy due to ignorance of it. Only one of these cases failed to respond and required surgical excision, which failed to save the patient.

Roentgen rays of 127–150 kv. with 0 to 3 mm. Al were employed, depending on the depth of the lesion. From 200 to 250 r were applied as the initial dose and from 100 to 150 r in a day or two if proper reaction had not occurred.

The usual course of events were intensification of pain and elevation of temperature two to three hours after treatment, followed by marked relief of pain within a few hours, disappearance of induration, and sharp localization of the lesion in from 24 to 48 hours, drainage through a single opening, and general subjective improvement. The acute inflammatory phase of the lesion usually subsided entirely in about two weeks and the resultant scar was very slight—some were completely aborted.

This treatment should be instituted as early as possible. In every case the patient is kept under surgical observation for other steps, when necessary—usually, only for very slight incision.

S. M. ATKINS, M.D.

DIATHERMY

Deep Effect and Localization in the Short Wave Condenser Field. F. Nagelschmidt. British Jour. Radiol., July, 1935, **8,** 449–456. (Reprinted by per-

mission from British Med. Jour., Sept. 21, 1935, p. 45 of Epitome of Current Medical Literature.)

Prominent difficulties in the technic of short wave therapy are, according to this author, that the condenser field suffers deformation by the introduction of any object into the field between the two electrodes; the intense radiation which emanates in all directions from the electrodes, the wires, and the patient, and the absence of any practical measuring device. He believes that he has been able to overcome the first two of these sufficiently for practical purposes. He interposes suitably shaped auxiliary dielectric electrodes in order to concentrate the majority of the power of the whole field energy to a desired part of the portion of the body to be irradiated. Contact with the auxiliary electrodes (with or without a small air-space between the metallic pole plates) and the dielectrics reduces considerably radiation losses by its damping effect. The size of the metallic pole plates can be relatively small. The dielectric employed (a glass tube containing white of egg) brings about an exact localization of the heating. When the size or form of the part to be irradiated is unsuitable, the difficulty can be surmounted by the introduction of auxiliary electrodes of wax-paraffin mixtures or agar with certain admixtures.

DOSAGE

The Testing of the Calibration of Small Chamber Dosage Meters. G. Spiegler and K. Juris. Strahlentherapie, 1935, **54,** 174–181.

The authors found that tests used so far for the calibration of small ionization chambers do not satisfactorily detect variations due to changes in atmospheric pressure and temperature. Errors in dosage may be as high as 10 per cent. They constructed, therefore, a new standard of comparison based on beta radiation of radium, which indicates changes proportional to fluctuation in pressure and temperature.

ERNST A. POHLE, M.D., Ph.D.

ENDOCRINE GLANDS

Roentgen Examination in Endocrine Pathology. J. Morelle and H. Solé. Jour. de Radiol. et d'Electrol., July, 1935, **19,** 360–400. (Reprinted by permission from British Med. Jour., Sept. 21, 1935, p. 45 of Epitome of Current Medical Literature.)

The authors review comprehensively the part which radiology may play in elucidating diseases of endocrine etiology. Thus diminution of the normal transparency of the pituitary suggests calcareous impregnation, while granular concretions may be recognized in its fossa or changes in its immediate neighborhood affecting the bone or brain. Early calcification may similarly be recognized in the pineal gland, but must be differentiated from calcareous changes in the choroid plexuses, which are generally bilateral and give ring-shaped shadows. Intra-parenchymatous calcareous deposits may also be seen in the thyroid gland, especially in adenomatous degeneration. Thyroid hypertrophy appears as a diffuse shadow, and retrosternal extension can be distinguished when present. Radiological examination during deglutition is particularly helpful. In hyperthyroidism and hypothyroidism the skeletal changes are discernible, while in the latter condition disturbances in the heart and digestive tract may be observed. Tumors of the parathyroid and suprarenal glands can also be detected.

THE EYE

Roentgenograms of Calcareous Cataracts. Eugen von Engelmayer. Röntgenpraxis, August, 1935, **7,** 520–522.

The roentgenographic appearance of a calcareous cataract of the lens is described. The roentgen examination is able to differentiate between the different types of calcareous cataracts because it is able to demonstrate very fine details of structure.

HANS W. HEFKE, M.D.

FEVER THERAPY

Preliminary Study of the Effect of Artificial Fever upon Hopeless Tumor Cases. Stafford I. Warren. Am. Jour. Roentgenol. and Rad. Ther., January, 1935, **33,** 75–87.

There is *in vitro* (and some *in vivo*) experimental evidence suggesting a definite "thermal death time" for transplantable animal tumors at high febrile temperatures. There is a distribution of heat by the circulating blood in the febrile state, so that the internal organs and tumors in animals, and presumably in humans, reach and can be maintained at any predetermined temperature level. This is usually nearly equal to the rectal temperature.

Upon this basis, 32 hopelessly advanced human cases of different types of malignant disease were treated by generalized fever therapy, and by a combination of fever therapy at 41.5° C. and deep roentgen therapy.

In cases previously treated by deep roentgen therapy, and in untreated cases, fever therapy seems to have a definite destructive effect upon the tumor cells. The amount of destruction and its duration vary from case to case. Further work on this aspect of the treatment of malignant disease seems to be warranted in the hope of obtaining an additional aid in the treatment of this malady. It should be emphasized that no results approaching cure have yet been obtained by this procedure.

S. M. ATKINS, M.D.

GASTRO-INTESTINAL TRACT (DIAGNOSIS)

Roentgenologic Determination of Normal and Abnormal Results Following Operation on the Stomach and Duodenum. B. R. Kirklin. Am. Jour. Roentgenol. and Rad. Ther., April, 1935, **33,** 468–480.

There is no essential difference in the roentgenologic technic of post-operative from pre-operative examination of the stomach; however, the barium meal should

be administered more slowly in order that adequate study of the mucosal pattern and operative stoma may not be obscured.

When the first post-operative study is made shortly after surgery has been performed the stomach is apt to be found atonic and containing more or less secretion in the region of the new stoma. There is apt to be more or less irregularity, probably due to tissue-swelling, and the passage of the opaque material through the new opening is apt to be slower than will be observed later on. Even after many years the stoma does not tend to close except as the result of marginal ulcers or other diseases. On the other hand, if the pylorus was blocked by sutures it will not remain closed but usually reopens within a few weeks.

The roentgenologic findings immediately and remotely following surgery are described for the various more common gastric operations including Billroth I, Billroth II, sleeve resection, local resection, total gastrectomy, pyloroplasty, gastroduodenostomy, entero-enterostomy, both in cases in which the desired results are obtained and in certain instances wherein complications and abnormal results follow.

Concerning obstruction occurring after operation, the following causes may be the basis for the disturbed physiology: kinking of jejunal wall opposite stoma; prolapse of gastric mucosa into stoma; closure of the stoma; acute angulation at the duodenojejunal juncture, constriction of jejunum by contraction of opening in transverse mesocolon, volvulus of the efferent jejunal segment, hernia of the jejunum through the opening in the mesocolon, and intussusception of the jejunum into the stomach.

Errors in surgical technic have not been commonly encountered by the author. There is, of course, a fairly wide range in determining size and location of the stoma and length of the proximal jejunal segment, which should be neither too short nor too long.

J. E. Habbe, M.D.

GENITO-URINARY TRACT (DIAGNOSIS)

Rupture of the Urinary Bladder. Louis Berger. Am. Jour. Surg., September, 1935, **29,** 460–462.

The author reports a case of rupture of the bladder. From the history and findings of his case one is led to speculate as to the cause of the rupture. The question arises as to the possibility of an incomplete rupture occurring at the time of the injury; namely, the leaving of all coats except the peritoneum, which holds the bladder intact until, at some future time, coughing or over-distention may give rise to a tear in the peritoneum itself, with subsequent complete rupture.

Davis H. Pardoll, M.D.

GRENZ RAYS

Problems in Grenz-ray Therapy. Friedrich Kalz. Strahlentherapie, 1935, **54,** 96–113.

The author undertook a very detailed study of the skin reactions following exposure to Grenz rays under a large variety of conditions. He found that the reaction occurs in cycles as described before by Miescher for ordinary roentgen rays. Following doses of from 500 to 1,500 r the erythema usually occurs in three cycles with the peak in the third cycle. Fractional application of the dose reduces intensity and duration of the skin reaction even after doses of from 600 to 1,000 r. Some cases show erythema with a bluish hue indicating a late injury of the blood vessels. Occasionally there occurs a so-called "early erythema" which is quite different from the usual erythema. Hyperemia of the skin brings out the irradiated area even years after the exposure. The article is well illustrated.

Ernst A. Pohle, M.D., Ph.D.

HEART AND VASCULAR SYSTEM

The Clinical Value of Cardiac Measurements in Dorsoventral and Oblique Projections. J. G. Dillon and J. B. Gurewitsch. Fortschr. a. d. Geb. d. Röntgenstrahlen, 1935, **51,** 180–190.

A discussion of the various important methods of cardiac measurements, advocating the procedure of Walter Fray (Am. Jour. Roentgenol. and Rad. Ther., 1932, **27,** 177, 585, 729), as it permits of estimation of the size of each ventricle.

H. A. Jarre, M.D.

THE KIDNEYS

Confusing Clinical Manifestations of Malignant Renal Neoplasms. C. D. Creevy. Archiv. Internal Med., June, 1935, **55,** 895–916.

The first symptom of a malignant neoplasm of the kidney is often due not to the primary tumor itself, but to a distant metastasis or to extension to a neighboring organ. Such a metastasis or extension may mimic a neoplasm of the bone, pulmonary disease, a lesion of the brain, spinal cord or peripheral nerves, cirrhosis of the liver, a lesion of the stomach or colon, or a tumor of the neck, or it may cause unexplained anemia, fever, or cutaneous lesions. It may also cause confusion with other diseases of the urinary tract or be masked by them. The primary tumor may be mistaken for an ovarian cyst. An additional case is reported in which the diagnosis was not made during life because of an associated undulant fever.

The lesions are best recognized in obscure cases by bearing in mind the possibility of a renal tumor, eliciting an accurate history and subjecting the patient to a careful physical examination, then excluding or ascertaining that diagnosis by intravenous urograms, supplemented, as is often necessary, by retrograde pyelograms.

There was no instance in this group of solitary bony metastasis. Thirty-six different organs—counting bones as one organ system—were involved by metastasis in 92 cases of malignant renal neoplasms reviewed. Twelve different bones were affected.

E. M. Shebesta, M.D.

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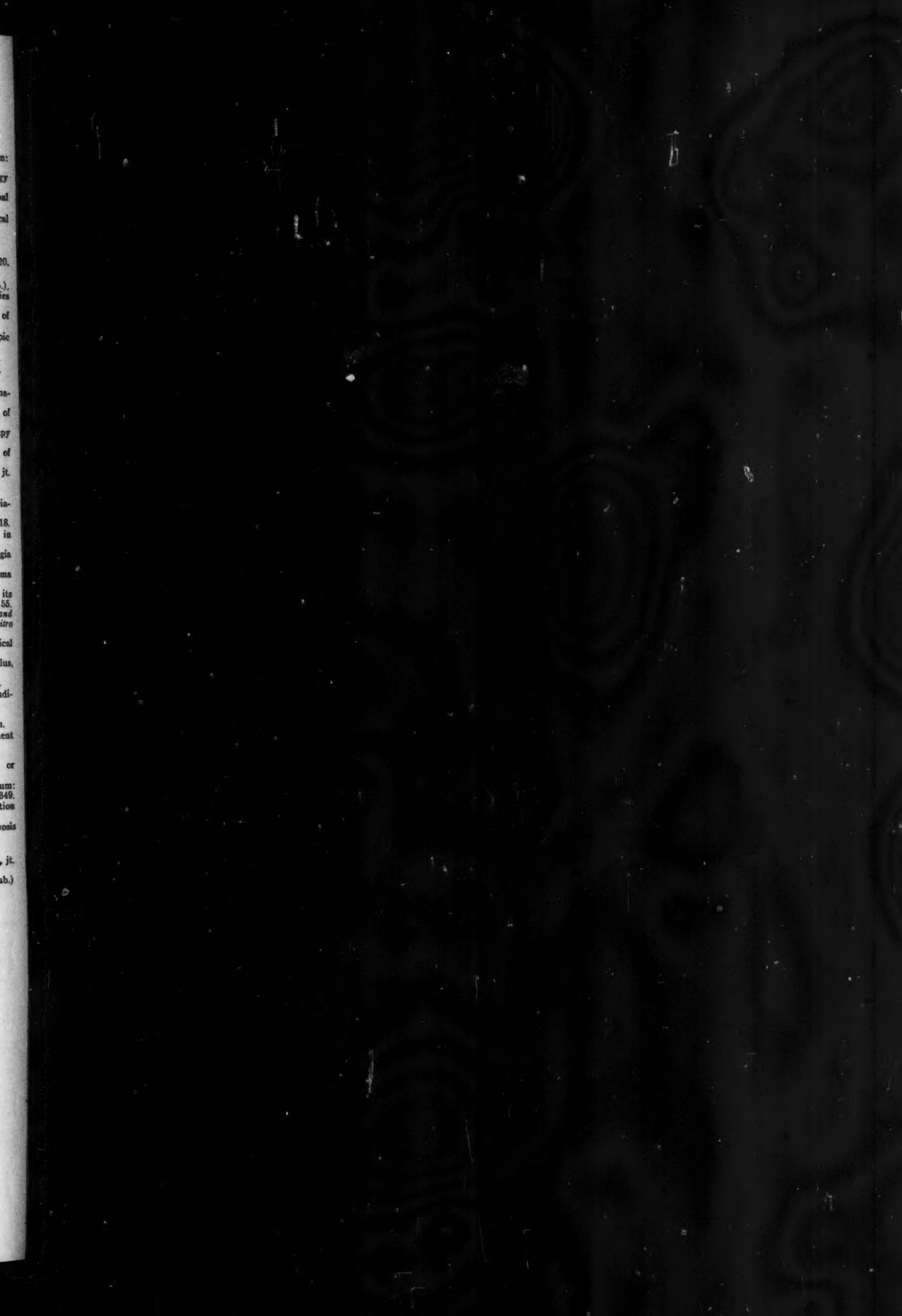
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